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PULMONARY VALVULOTOMY: WITH OR WITHOUT CARDIOPULMONARY BYPASS?*

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Many of the cardiac lesions amenable to surgical therapy can be treated in only one basic manner. Extracardiac congenital disorders such as patent ductus arteriosus and coarctation of the aorta are managed well by "closed" techniques, and adjuvants such as hypothermia with inflow occlusion or cardiopulmonary bypass during extracorporeal circulation are not applicable. Such lesions as ventricular septal defects, in contrast, cannot be managed satisfactorily by any method except open cardiotomy with total cardiopulmonary bypass. On the other hand, certain abnormalities of the heart, such as pulmonary valvular stenosis, aortic and subaortic valvular stenosis and tetralogy of Fallot, can be treated either by "open" or "closed" methods. Careful assessment of the advantages and disadvantages of both procedures is necessary in order to decide which is preferable, whether either should be utilized in all cases, and, if not, the indications for each. The present study is an effort to reach such decisions with reference to congenital pulmonary valvular stenosis with an intact ventricular septum and the lesions which may be associated with it. Here the problem is even more complex, since open valvulotomy can be accomplished with inflow occlusion in the normothermic or hypothermic state, with right heart bypass employing extracorporeal circulation, and with total cardiopulmonary bypass and extracorporeal circulation.

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REVIEW OF METHODS

Closed valvulotomy. In 1948 Sellors[20] and Brock[3] each described the procedure of closed transventricular instrumental division of the stenotic pulmonary valve and during the next few years Brock rapidly acquired a rather large experience with this approach in the treatment of pulmonary valvular stenosis occurring alone or as a part of the tetralogy of Fallot.[4, 5] Brock's valvulotome became widely used. New valvulotomes were described by Potts and his associates[19] and by Muller and Longmire.[17] Initial experiences indicated two deficiencies of this operative procedure. In the first place, performance of the valvulotomy through the left anterior intercostal approach as Brock had originally recommended was often attended by some difficulty in exposure of the anterior surface of the right ventricle and, more important, by frequent difficulties with rhythm and cardiac function during the necessary manipulations—a circumstance which, as Blalock and Kieffer[1] pointed out, often made it necessary to hurry with the valvulotomy itself and sometimes resulted in a fatal outcome. Such troublesome and frightening occurrences were eliminated by exposure through the median sternotomy incision which we began to employ in 1951.[21] In the second place, it soon became evident from postoperative catheterization studies that passage of the available valvulotomes and dilators through the stenotic valve often stretched and dilated the stenotic orifice without incising it in such a way as to relieve the constriction adequately. This was evident from the postoperative catheterization studies of Lurie and Shumacker,[14] Kirklin and his associates,[11] Humphreys and his

colleagues,[10] Ekström and his co-workers,[7] Blount and his group[2] and others, all of which showed failure of right ventricular pressure to return to normal in the majority of cases. Even those who felt that the outcome was good had imperfect results. For example, Lillehei and his colleagues[13] found on postoperative catheterization that one-fourth of their patients had no reduction in right ventricular pressure, although the pressure had fallen to an average of 43 mm. systolic in the remainder. It became evident that the valvulotomy had to be performed in a different manner. Since September 1954 we have utilized a Kerrison rongeur after passage of the Brock valvulotome. The rongeur is positioned until its opened jaws are in the region first of one and then of the second area of the primary incision. The jaws are then closed so as to engage the remaining uncut portion of the valve back to its point of union with the pulmonary artery itself. One can make sure of this position by gentle traction upon the closed instrument which, when it is properly placed, indents the adjacent arterial wall. A narrow strip of valve substance is thus removed to complete the wide valvular incision.

Open valvulotomy with hypothermia and inflow occlusion. Open valvulotomy under moderate hypothermia and venous inflow occlusion was introduced in 1953 by Swan.[26] It was soon evident that this method permitted a more complete surgical relief of the stenosis as evidenced by postoperative catheterization studies and that it it could be accomplished with a low mortality.[2, 22, 23, 25, 26] Its chief disadvantage lay in the limited period for working with the pulmonary artery open and, more particularly, for safely correcting associated lesions such as atrial septal defect and infundibular stenosis.

Open valvulotomy with inflow occlusion in the normothermic state. In 1959 Lam and Taber[12] demonstrated that it was possible to perform open pulmonary valvulotomy safely in the normothermic state with low mortality. As far as I know, others have not used this method since the safe period of inflow occlusion is even briefer than when moderate hypothermia is employed.

Open valvulotomy with right heart bypass and extracorporeal circulation. In 1953 Dodrill[6] performed an open pulmonary valvulotomy during right heart bypass with extracorporeal circulation. As far as I know, this was the first successful use of the pumping portion of the heart-lung machine for an open operation, preceding by about six months Gibbon's[9] successful correction of an atrial septal defect with total cardiopulmonary bypass. Apparently this technique has not been used subsequently for pulmonary valvulotomy.

Open valvulotomy with total cardiopulmonary bypass. In 1958 McGoon and Kirklin[16] reported a good outcome in 10 cases of pulmonary valvular stenosis, many complicated by associated infundibular stenosis, utilizing extracorporeal circulation and total cardiopulmonary bypass.

CLINICAL MATERIAL AND RESULTS

Altogether we have treated by operative valvulotomy 78 patients with pulmonary valvular stenosis and presumably an intact ventricular septum (table 1). Since some of the early ones were not studied by cardiac catheterization or cineangiography, it is possible that a few may be included in whom there is a ventricular septal defect. All patients, however, in whom this associated lesion is known to have been present have been excluded from this study.

Closed valvulotomy. There were 46 patients treated by closed valvulotomy. Of these, 5 were emergency procedures upon small, very ill, cyanotic infants. Their ages were 6, 10, 12, 15 and 16 months. All were in failure and had hepatic engorgement; 3 were rather markedly edematous; 1 was stuporous. None had preoperative catheterization. One, who appeared moribund, was rushed from the admitting room directly to the operating room. A second was picked up on the ward in an apparently nearly terminal state and literally carried on the run to the operating room. All survived and in all a remarkable improvement was noted as soon as the stenotic valve was opened. Edema and other evidences of failure disappeared in the immediate postoperative period. In 2, definite decrease in heart size was noted as early as the second day. All have done well.

There were 4 additional patients operated upon as urgent cases. Several had been studied by cardiac catheterization. They were all quite ill. Their ages were 5 months, 2, 2½ and 10 years. Improvement was striking as soon as the valve was opened. All had an uneventful recovery and have had a good result to date. One who was considered too ill for preoperative catheterization had an oxygen saturation before operation of 50

TABLE 1

Results of operative treatment of pulmonic valvular stenosis

Procedure	No. of Patients	Age in Years		Operation	Associated Lesions Corrected at Time of Operation	Deaths
		Range	Average			
Closed transventricular valvulotomy	5	0.5–1.3	0.8	Emergency	None	0
	4	0.4–10	3.7	Urgent	None	0
	37	0.7–37	13	Elective	One with secundum atrial septal defect	0
Open valvulotomy with cardiopulmonary bypass	19	5–57	18.3	Elective	None	1
	3	7–12	10	Elective	Muscular outflow obstruction	0
	2	13–18	15.5	Elective	Infundibular stenosis	0
	4	4–10	6.4	Elective	Secundum atrial septal defect	1
	1		5	Elective	Secundum atrial septal defect; partial anomalous pulmonary venous drainage	0
	2	8–10	9	Elective	Secondum atrial septal defect; muscular outflow obstruction	0
	1		5	Elective	Septum primum atrial septal defect; cleft mitral valve	0

per cent and a hematocrit of 83. Postoperatively the right ventricular pressure was found to be 33/3 and the oxygen saturation 95 per cent when the child was breathing room air. He had a normal cardiac output at rest and a normal response to exercise.

We had 37 patients, ranging in age from 8 months to 37 years, who have undergone elective closed valvulotomies. In addition, 1 had a secundum type atrial septal defect which was repaired at the same time. Of this group, 6 were 5 years or less in age, and 19 were between 6 and 15. The remainder were older; 3 were in their 30's. All survived and had an initially good result. There have been 3 lost to follow-up. Of the remaining 34 all have done well clinically except 1, a 14-year-old girl who was originally operated upon in 1952 at the age of 5. The valve was felt to be extremely tough, thick and possibly calcified. The right ventricular pressure was 147/5 preoperatively, 120/8, 6 months afterwards. She was clinically improved until 1959 when she noted recurrence of fatigue with exercise. On recatheterization in 1960 the right ventricular pressure was 179/11. She has recently had a successful open repair with cardiopulmonary bypass.

These patients include 6 in whom there was clear-cut cineangiographic evidence of a diminutive ventricle. They all survived and did well.

The first 6 patients were operated upon through a left anterolateral incision. In 4, alarming transient rhythm disturbances associated with poor cardiac function occurred before valvulotomy. Such difficulties have not been observed in the remaining patients in whom a sternal splitting incision has been used. The first 32 patients were treated by passage of the largest possible Brock valvulotome through the stenotic valve and then by dilation, chiefly accomplished by spreading the blades of a curved hemostat. After passage of the Brock instrument, a small segment of the valve was actually excised back to the point of attachment to the pulmonary wall with a Kerrison rongeur in the last 14 people. This was done bilaterally to complete the incision of the stenotic valves. In recent cases the mattress suture placed in the ventricular wall for control of bleeding has been supported by strips of Teflon felt, a maneuver we find very useful. Also, in recent cases the opening in the ventricular wall has been made according to the suggestion of Wooler[37] for creating a pathway through the ventricular apex in performing instrumental closed mitral valvulotomy. The epicardium is incised with a small knife. Hegar dilators are passed through the ventricular wall in gradually increasing sizes and then the appropriate valvulotome is introduced. This maneuver seems to split

muscle fibers rather than cut them and makes control of blood loss much easier. After completion of the valvulotomy, the pericardial incision is left open and drainage is established either with a mediastinal catheter when hemostasis is particularly secure, or by opening the right pleural cavity and introducing into it a drainage tube through an intercostal stab wound. The sternum is closed with wire sutures, the rest of the layers with interrupted nonabsorbable material.

The results, as far as immediate adequate reduction of right ventricular pressure, have been much better since the newer technique of valvulotomy has been utilized. We have studied 12 patients treated by the earlier method postoperatively by cardiac catheterization (table 2; fig. 1). They were selected more or less at random for this study. Relatively good reduction in right ventricular pressure has been found in half of them. In the remainder the postoperative right ventricular pressure ranged from 60 to 120.

TABLE 2

Pre- and postoperative catheterization data

Case No.	Date of Operation	Interval between Operation and Postoperative Catheterization in Years	Right Ventricular Pressure in mm. Hg		Pulmonary Artery Pressure in mm. Hg		RV-PA* Gradient		
			Preop.	Postop.	Preop.	Postop.	Preop.	Postop.	Per Cent Reduction
Early Closed Valvulotomy									
1	April 1951	1.5	120/0	101/0	2/–2	3/–2	118	98	17
2	Sept. 1951	1	105/9	106/0	25/16	34/11	80	72	10
				96/7		27/6		69	14
3	Sept. 1951	0.7	65/–7	39/–5	10/–1	12/2	55	27	51
4	Sept. 1951	1	86/–1	43/2	45/5	28/19	41	15	63
5	Sept. 1951	3		33/3		13/2		20	
6	Oct. 1951	0.5	57/0	48/6	24/11	20/8	33	28	15
7	Dec. 1951	0.3	127/–7	84/–3	1/0	14/4	126	70	44
8	Mar. 1952	0.5	147/5	120/8	17/–3	20/10	130	100	23
		8.5		179/11		9/0		170	
9	May 1953	0.25	110/2	75/5	24/7	40/2	86	35	59
10	May 1953	1	154/–1	60/6	24/7	15/1	130	45	65
11	May 1954	5.5	157/5	38/5	31/11	19/7	126	19	85
12	May 1954	5	36/5†	23/4	18/10	18/7	18	5	72
Later Closed Valvulotomy									
13	Jan. 1955	0.75	223/–3	57/–3	7/	22/–4	216	35	84
14	Nov. 1957	0.2	160/5	50/4	14/8	15/7	146	35	75
		1		43/5		13/7		30	80
15	May 1959	1	163/3	35/1	0	16/0	163	19	88
Open Valvulotomy									
16	Mar. 1958	2.5	207/3	36/5	–43/–7	23/0	250	13	95
17	May 1958	0.5	105/7	54/12	18/5	24/10	87	30	66
18	June 1958	1.7	176/12	45/7	–10/–2	22/6	186	23	88
19	Oct. 1959	1.2	200/0	28/–5	0	12/2	200	16	92

* RV, right ventricular; PA, pulmonary artery.

† Under anesthesia.

When the pulmonary artery could not be catheterized its pressure was assumed to be zero in calculating RV-PA gradient.

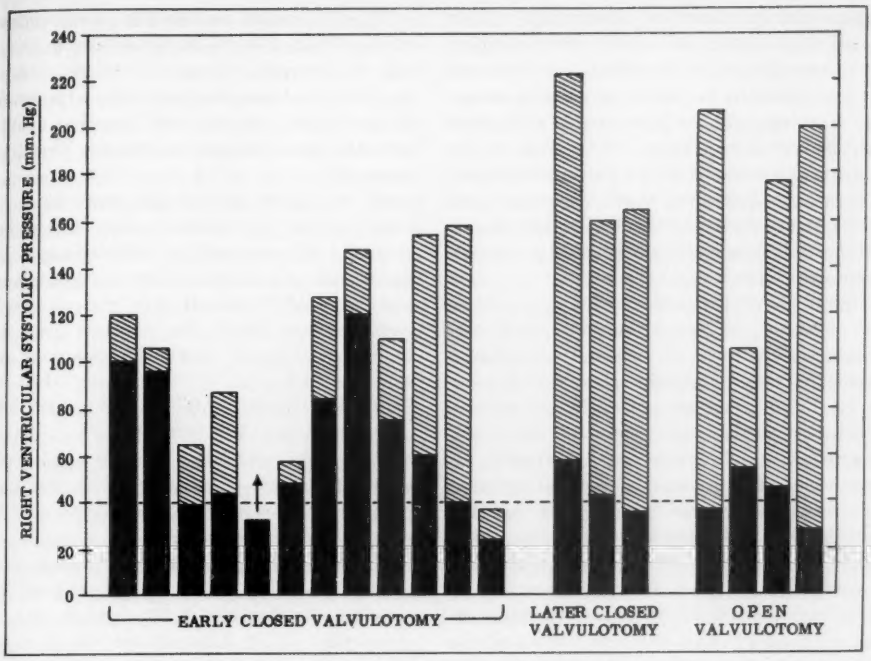

FIG. 1. Comparison of right ventricular systolic pressures before and after pulmonary valvulotomy. The *height* of the total column represents the preoperative pressure, the *shaded area* the postoperative, and the *crosshatched* area the reduction in pressure.

One of the patients who had return of symptoms after a number of years and was found to have a residual high right ventricular pressure, has, as previously mentioned, been recently reoperated upon successfully by the open technique. It is our plan to recatheterize all of the patients. It is obvious that if no further improvement has occurred in a number of them, reoperation is in order. Postoperative catheterization has been carried out in only 3 of the patients treated by the later closed technique. They were all patients in whom there was a significant degree of muscular outflow obstruction, and a failure of significant reduction of ventricular pressure at the time of operation when such measurements were made. The ventricular pressure had fallen from 223 to 57, 9 months after operation 1, from 160 to 43 a year after operation in another, and from 163 to 35 a year after surgery in the third. As far as we can tell at the present time, it seems unlikely that any patient operated upon by the later method will require reoperation.

Open valvulotomy with cardiopulmonary bypass and extracorporeal circulation. There were 32 patients, ranging in age from 4 to 57 years, who had open valvulotomies with total cardiopulmonary bypass and extracorporeal circulation. In 19 nothing was done at the time of operation except the valvulotomy. Some had hypertrophied muscle in the ventricular outflow tract causing some obstruction, but the response to valvulotomy was considered satisfactory and it was felt that the muscular obstruction would resolve later. In 3, with no other complicating defect, there was, in addition to the valvular obstruction, a high degree of muscular outflow obstruction and a persistent high ventricular pressure after valvulotomy. The outflow tract of the right ventricle was enlarged in 1 patient by resection of muscle tissue and in the other 2 by insertion of a patch into the ventriculotomy incision. Two patients had true infundibular stenosis in addition to the valvular stenosis and both lesions were repaired. In 1, a patch was inserted into the ventriculotomy incision. Of our patients 7 had secundum type atrial septal defects, the so-called trilogy of Fallot;[24] 1 of them also had partial anamolous pulmonary venous drainage;. 2 had

marked muscular outflow obstruction and required the insertion of a patch into a ventriculotomy incision. All of the defects were corrected in these patients. In addition, a ninth patient had a septum primum defect and a cleft mitral valve, which were repaired at the time of pulmonary valvulotomy. All of these patients survived except 2. Both of these exceptional cases had a small ventricle with hypertrophied ventricular wall, causing the syndrome of generalized obstructive ventricular hypertrophy.[15]

One of the 2 was a 10-year-old girl who had had cyanosis, exercise intolerance and slow growth since infancy. At preoperative catheterization the right ventricular pressure was found to be 196/6 when the systemic pressure was 87/68. Cineangiograms showed a small right ventricle with diffuse muscular hypertrophy and evidence of an atrial septal defect. At operation the stenotic valve was opened along the fused commissures and the secundum type atrial septal defect was closed. In an effort to relieve the outflow obstruction, an incision was made in the distal portion of the right ventricle and through the pulmonary ring and a patch of compressed Ivalon sutured in place. After a reasonably satisfactory period, she suddenly became hypotensive and unresponsive the night of operation. No benefit resulted from the use of vasopressors or transfusion. In order to eliminate the possibility of tamponade, the wound was reopened, but poor cardiac function continued and during closure of the wound, arrest occurred and resuscitation could not be achieved. The postmortem examination confirmed the presence of a diminutive right ventricular cavity. The other child was 9 years of age. Although he was in congestive failure preoperatively, a rather good response followed digitalization. Cineangiograms revealed the right ventricle to be quite large in comparison with the small left ventricle. Following valvulotomy the right ventricular pressure fell from 180/0 to 90/0, systemic pressures being 100 and 90 respectively at the time of the two measurements. Shortly after operation he developed pulmonary edema and died during the night. The postmortem examination confirmed the small volume of the left ventricular cavity, and it was felt that the cavity was unable to handle the sudden increase in volume flow resulting from the increased circulation through the pulmonary vascular bed.

Thus all patients recovered from valvulotomy, whether treated by the closed or open technique, with the exception of these 2 children with the syndrome of obstructive ventricular hypertrophy. Of particular interest is the fact that 6 others with the same difficulty survived a closed valvulotomy.

All the surviving patients have done well following open valvulotomy. Only 4 have been studied postoperatively by catheterization. One had a near normal right ventricular pressure and a reduction of 171 mm. Hg from the preoperative catheterization level. In another, who had improved clinically and had undergone considerable reduction in heart size, the right ventricular pressure was 54, 6 months after operation. A third who had not had a satisfactory drop in right ventricular pressure immediately after valvulotomy—a situation which would now prompt us to increase the outflow tract of the right ventricle by insertion of a patch—was found to have a right ventricular pressure of 45, a little over 1½ years after operation. This constituted a decrease of 131 mm. Hg from the preoperative level. A fourth patient, 14 months after operation was found to have a ventricular pressure of 28, in contrast to 200 preoperatively. She was a 15-year-old girl who had been treated in another institution by closed valvulotomy with an unsatisfactory result.

The operative technique briefly was as follows: A rotating disk oxygenator was used. The inferior and superior vena caval blood was drained from the patient by gravity. The arterial inflow was introduced through a cannula in the femoral artery. All except the last patient were operated upon in a moderately hypothermic state. In order to eliminate the lag in fall of muscle and brain temperature which occurs with extracorporeal cooling of a patient, an ice bag was placed under the patient shortly after induction and removed after the cannulations were performed. The temperature was then further reduced by a heat exchanger to from 30° to 32° and, after completion of the operative procedure upon the heart, the patient was rapidly rewarmed to 35°C. We shall probably operate upon patients without hypothermia in the future. In the earlier cases the pulmonary valve was exposed through a vertical incision in the pulmonary artery. The more recent cases have had exposure of the valve through a transverse incision in the pul-

monary artery and we are convinced that this approach provides far better visualization of the valve. The valves were opened under good vision either with a scissors or a scalpel. A finger was then passed through the valve and the pulmonary ring in order to make sure there was no residual obstruction and to detect any unusual degree of ventricular outflow obstruction during ventricular systole. When an outflow patch was required in the earlier cases, an oval piece of compressed Ivalon was sutured into a vertically placed incision. It is now our custom to utilize instead a free pericardial graft covered with a thin piece of Teflon felt.

PRESSURE MEASUREMENTS AT THE TIME OF OPERATION

We have been impressed by the frequency with which right ventricular and pulmonary artery pressures measured at the time of operation were substantially lower than those measured preoperatively by catheterization. Analysis of a number of patients in which such data were recorded (figs. 2 and 3) shows that this discrepancy occurred predominantly in the group operated upon with extracorporeal circulation and moderate hypothermia. The prevalvulotomy pressures were measured after the cannulations were carried out, and although the precise temperature of the patient at the time was not recorded, the average esophageal temperature was approximately 33 or 34°. It is known that the cardiac output of anesthetized dogs at a body temperature of 33° is less than 70 per cent that at 37°C. It is probable that the decreased cardiac output in the somewhat hypothermic state is largely responsible for these discrepancies. It will be seen in figure 2 that only in 3 of 17 patients was there reasonable agreement between the right ventricular-pulmonary artery gradient preoperatively and at the time of operation. The correlation was much better in those patients treated by closed valvulotomy and in the single patient treated by cardiopulmonary bypass at a relatively normal body temperature. In 6 of these 9 patients there was reasonably good correlation between the right ventricular-pulmonary artery gradient at the time of operation and during preoperative catheterization. In spite of the frequency with which the gradient has been less at operation than preoperatively, measurements of pressure before and after valvulotomy probably revealed a significant trend. This is particularly likely since, in the hypothermic patients, the postvalvulotomy pressures were measured when the esophageal temperature was 35°C. Because

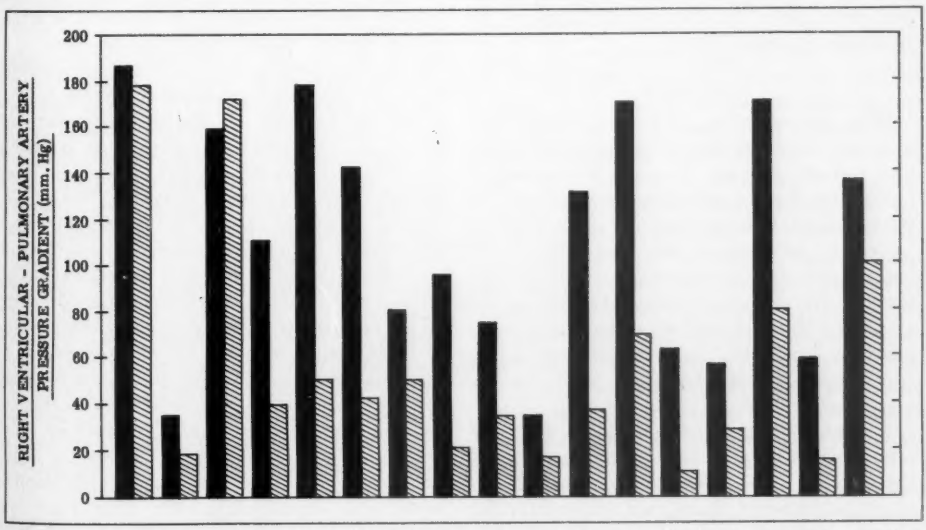

FIG. 2. Comparison of right ventricular-pulmonary artery pressure gradients at preoperative catheterization and at time of surgery in a group of patients treated by open valvulotomy during cardiopulmonary bypass in the moderately hypothermic state. *Solid columns*, preoperative; *crosshatched columns*, operative gradients.

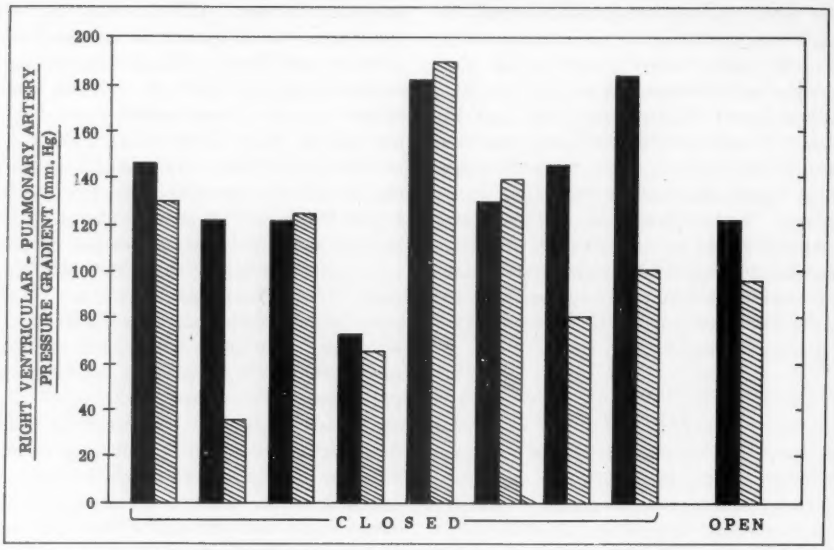

FIG. 3. Comparison of right ventricular-pulmonary artery pressure gradients at preoperative catheterization and at time of surgery in patients treated by closed valvulotomy or by open valvulotomy during cardiopulmonary bypass in a normothermic state. *Solid columns*, preoperative; *crosshatched columns*, operative gradients.

the final pressures were measured at a higher body temperature, any observed decrease in right ventricular pressure or in ventricular-pulmonary artery gradient would appear to be all the more meaningful.

DISCUSSION

With the exception of the 2 patients who underwent an open pulmonary valvulotomy and who had the peculiar syndrome of ventricular obstruction due to generalized hypertrophy of the ventricular wall and small size of the ventricular cavity, all patients recovered. This would suggest that with the exception of patients presenting this unusual complication, both the closed and the open methods of treatment carry relatively small risk. In attempting to decide which approach is preferable, other matters must be considered.

The argument may be advanced that the open procedure permits a better pulmonary valvulotomy. It is evident from our own experiences and those of others that transventricular closed valvulotomy as it was originally performed often ended in inadequate relief of the pulmonary valvular stenosis. It would appear equally evident that the newer technique of closed pulmonary valvulotomy, in contrast, results in complete division of the stenotic valve and there is every reason to believe that it results in relief of the valvular obstruction. It is also clear that the open approach, particularly when carried out with total cardiopulmonary bypass which allows the operator all the time necessary to incise the valve as accurately as is possible, inevitably relieves the valvular obstruction. It is unlikely that any patient treated by the closed method fails to have some resultant pulmonary insufficiency. This is a reasonable assumption since the incisions in the valve leaflets are made without the benefit of direct vision. On the other hand, we know of no patient in our own series, or in any other, who has as yet developed any symptoms due to pulmonary insufficiency. Only further observation will, however, determine whether these patients will go through life without related symptoms. Furthermore, it is almost positive that the vast majority of patients have some insufficiency even after carefully performed open valvulotomy. Occasionally, the stenotic pulmonary valve is found to be a thickened, fibrosed and sometimes calcified

structure with no visible commissural fusion and with no adequate valve sinuses. As one incises such valves, he gains the impression that competent function afterwards would be almost impossible. On the other hand, one occasionally meets with a stenotic pulmonary valve in which the three leaflets are clearly discernible, fused together, each having a reasonably well developed valve sinus. Undoubtedly, open valvulotomy in such cases should result in fairly good competency. The vast majority of the congenitally stenotic pulmonary valves are elongated, dome-shaped valvular structures with a central rounded orifice. The surgeon cannot distinguish one leaflet from another nor any commissural fusion in the peripheral portion of the valve. However, in practically all such cases there are shallow valve sinuses at the base and short ridges which represent points of commissural fusion of the 3 leaflets. It is not likely that these valves will be entirely competent even when opened under direct vision, and yet the chance of this occurring is infinitely better with the open than with the closed procedure. In summary, it would be our feeling that the open operation has a slight advantage over the closed with regard to the valvulotomy itself.

Arguments might also be advanced in favor of the open operation, and particularly that done under total cardiopulmonary bypass and extracorporeal circulation, with respect to the discovery and correction of associated cardiac defects. Associated defects should not, however, be discovered at the time of operation but by careful study beforehand. The utilization of cineangiographic methods in conjunction with cardiac catheterization should clearly establish the presence of any one of the commonly associated abnormalities, atrial septal defect, ventricular septal defect, infundibular stenosis, muscular outflow obstruction, and the presence of a diminutive ventricle. It is true that a small, well guarded foramen ovale may occasionally be missed. It is generally agreed, however, that it does not require surgical repair and usually closes on its own after relief of the valvular obstruction and the right ventricular hypertension. With cardiac catheterization alone, a ventricular septal defect was occasionally not detected, particularly when right and left ventricular pressures were approximately the same and only a relatively insignificant bidirectional shunt was present. Such defects, however, should be discovered on cineangiography. As far as the treatment of any associated defect is concerned, the advantages definitely lie with the open procedure and total cardiopulmonary bypass. One can take care of them in an unhurried fashion. The specific problem of muscular outflow obstruction certainly requires further study. From the reported observations and from a few experiences of our own, it would seem likely that with the passage of time after an adequate valvulotomy this hypertrophy tends to resolve with return of ventricular pressures to normal or near normal levels. Swan has stated that this has invariably been true in his own experience.[22] On the other hand, Gerbode and his associates[8] have the conviction that failure to relieve such outflow obstruction at the time of pulmonary valvulotomy may result in a fatal outcome. So convinced are they that this is true, that they now recommend approaching the pulmonary valve through a ventriculotomy incision instead of through the pulmonary artery. It is our own feeling that the latter is the preferable approach. Indeed, in the operative repair of tetralogy of Fallot associated with valvular stenosis, we are inclined at the present to favor an additional pulmonary artery incision since the valve can be opened so much more accurately through this approach than by inverting it into a ventriculotomy incision. We have the feeling that after the pulmonary valve is opened, one should try to estimate the degree of muscular outflow obstruction by introducing a finger through the valve and the pulmonary ring. In some cases it is evident that there remains a high degree of obstruction since the outflow tract of the right ventricle contracts down forcibly upon the inserted finger. We also feel that one should measure pressures in the right ventricle and pulmonary artery before and after valvulotomy. If no substantial drop in right ventricular-pulmonary artery gradient occurs in such cases, we are inclined to make an incision in the ventricle and increase the size of the outflow tract by the insertion of a patch graft.

Taking all of these matters into consideration, we currently hold these views: The open operation is preferable in the vast majority of cases of pulmonary valvular stenosis with an intact ventricular septum. We are now inclined to

perform it with total cardiopulmonary bypass in the normothermic state. We feel that the closed method is probably preferable in certain circumstances. One is in the case of the very ill infant or small child in whom the operation must be carried out as an emergency or as an urgent procedure. All of the patients we have treated in these categories have survived and have done well. There may not be universal agreement in this respect. Mustard and his associates[18] lost 1 of 3 infants treated by closed valvulotomy shortly after it was performed, and a second patient died some years later with a persistently stenotic valve. In contrast, all 10 patients operated upon in the first year of life under hypothermia and venous inflow occlusion survived. Gerbode,[8] on the other hand, performed urgent open valvulotomy in 11 infants and small children with the use of hypothermia and inflow occlusion and 5 of them died. Because we have had no mortality in the patients operated upon as emergency or urgent cases, and they have all done well afterwards, we are inclined to believe that the closed method should be recommended for such patients. We are also of the opinion that the closed approach should be used in those patients who have a diminutive hypertrophied left or right ventricle. The only two fatalities in our series were patients in this category operated upon by the open method using cardiopulmonary bypass. We have deliberately chosen the closed approach in another 6 patients with the same condition. In contrast, they all survived and have done well. It is our hope that if they can be brought through a closed procedure, some of the ventricular hypertrophy may gradually resolve and the small ventricular chamber increase to an adequate size.

It is evident that further experiences may modify our views. It is important that the results of pulmonary valvulotomy by different operative methods be analyzed from the standpoint of mortality, the successful management of associated defects, the clinical response, and carefully performed postoperative catheterization studies.

CONCLUSIONS

1. Analysis of experiences with 78 open and closed pulmonary valvulotomies for pulmonary valvular stenosis with intact ventricular septum reveals that both procedures carry small risk. The only two deaths followed open correction in cases with the syndrome of generalized obstructive ventricular hypertrophy.

2. At present the open procedure utilizing cardiopulmonary bypass is recommended for most patients because it permits a somewhat better valvulotomy and permits correction of any associated defect. The closed procedure would seem preferable when an emergency or urgent operation must be performed in a very ill infant or small child and in those patients with a diminutive right or left ventricle.

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BREAST DISEASE AND MALIGNANCY: REVIEW OF 4190 CASES

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In this paper are presented the 17-year (1940 to 1957) analyses of the incidence and clinical evaluation of breast diseases from the surgical standpoint. These are compiled from the pathologic records at Doctors Hospital, Washington, D. C., covering 4190 cases in the categories of "non-neoplastic diseases," "benign tumors" and "malignant tumors."

The importance of these studies, as indicated by the numerous papers[2] on breast disease in the past 20 to 30 years, particularly from the standpoint of survival rate, have been stimulated by the intensive educational program for the laity. In this, patients were strongly alerted never to disregard any breast abnormality. This has certainly augmented the physician's responsibility to the patient, since there are no pathognomonic signs or symptoms by which all malignant growths can be recognized clinically, even though approximately 75 per cent of breast diseases fall into the category of non-neoplastic diseases or benign tumors. Thus the patient considers all breast abnormalities as potentially malignant until proved otherwise by adequate biopsy and frozen section study.

There is no question that the educational program in breast disease has been beneficial in that early cancer can be and has been determined with localization and curtailment of its spread. But whether or not the educational program has been overemphasized is a subject for discussion and deliberation, since in this day and time the management of breast disease has reached the point of crystallization.

METHOD OF STUDY

These records were slightly incomplete as to age during the war years. Cards were sent to the surgeons in attendance during the period from 1940 to 1957, requesting information as to therapy, recurrence, survival and related diseases. Due to the transiency of metropolitan Washington only 2084 follow-up cases were obtained. From these cases, clinical evaluations were made in the categories of non-neoplastic diseases, benign neoplastic diseases and malignant diseases of the breast. There was very little postoperative therapy except in the malignant diseases. This consisted of the standard irradiation therapy, which was used in 90 per cent of all the cases (table 1).

The category of non-neoplastic diseases is composed of chronic mastitis, abscess, fibrocystic disease, comedo mastitis, nodular fibrosis and fibrosing adenosis. Fibrocystic disease constitutes the majority of these cases, followed by fibrosing adenosis. A total of 835 patients with fibrocystic disease was studied. In these, the highest incidence of recurrence was in the 31 to 50 age group, with a preponderance in the 41 to 50 group. In 63 patients, among the 21 to 30 age group, 4 per cent developed benign tumors (fibroadenomas) within 5 years, while in the 51 to 60 age group, 3 per cent of 94 developed malignant tumors after 5 years. This occurrence of benign and malignant tumors is not considered to be related to the pre-existing breast disease.

Fibroadenomas were observed in 222 cases (table 2). Simple excision of these lesions served in the majority of cases to eliminate the tumor, although there were 14 per cent recurrences in the first 5 years, and 12 per cent recurrences in the 5- to 17-year group. Subsequent complications as a result of this entity were for all intents nonexistent. It was noted frequently from a pathologic point of view that the fibroadenomas in the older age group were commonly unusual patterns with variations from the commonly seen patterns in the younger age group.

Adenocarcinoma, intraductal carcinoma and Paget's disease, from the standpoint of incidence, compose the category of malignant tumors of the breast (table 3). Our study is limited to adenocarcinoma with local invasion and metastasis. It does not include intraductal

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papillary adenocarcinoma. On the follow-up, 319 patients were received. Of 38, among the 31 to 40 age group, 20 per cent died with metastasis within 5 years. The highest incidence of metastasis at the time was noted in this group. In the 41 to 50 age group, a similar percentage died within 5 years. It should be noted that the percentage with metastasis at the time of operation and the percentage dead and living with known metastasis, are quite similar; death from causes other than metastasis occurred more often after the age of 60. These figures compare favorably with those of other investigators as to the 5-year survival.

We agree with Dr. Heller of the National Institute of Cancer at Bethesda, Maryland, and Dr. Warren Cole,[1] Chicago, Illinois, that our statistics on the subject have become static and that in the next 2 or 3 decades we cannot expect to improve them unless we look beyond our past and present methods of treatment.

TABLE 1

Fibrocystic disease of breast in 835 patients

Age	No. of Patients	No Recurrence		Recurrence		Benign Tumors	Malignant Tumors
		1–5 yr.	5–17 yr.	1–65 yr	5–17 yr.		
yr.		%		%		%	
10–20	3	66	33	0	0	0	0
21–30	63	54	32	5	5	4	0
31–40	279	56	23	15	4	1.3	0.7
41–50	378	59	16	20	2	1	2
51–60	94	68	21	6	0	2	3
61–70	15	73	27	0	0	0	0
71–80	3	66	33	0	0	0	0
81–90	0	0	0	0	0	0	0

REMARKS AND CONCLUSIONS

From the clinical standpoint it has been fairly well established that non-neoplastic

TABLE 2

Fibroadenoma of breast in 222 patients

Age	No. of Patients	No Recurrence		Recurrence		No. of Patients with Non-neoplastic Breast Disease	No. of Patients with Benign Breast Tumor	No. Patients with Malignant Breast Tumor	
		1–5 yr.	5–17 yr.	1–5 yr.	5–17 yr.				
yr.		%		%					
10–20	8	63	37	0	0	0	0	0	
21–30	66	53	41	3	3	2	0	0	
31–40	100	45	38	11	6	4	1	1	(9 yr)
41–50	41	70	27	0	3	2	0	1	(7 yr)
51–60	6	83	17	0	0	0	0	0	
61–70	1	0	100	0	0	0	0	0	

TABLE 3

Adenocarcinoma of breast in 319 patients

Age	No. of Patients	Simple Metastasis	No. of Patients with Metastasis	Metastasis	Living with Unknown Metastasis		Living with Known Metastasis		Died with Known Metastasis		Died from Other Causes
					1–6 yr.	5–17 yr.	1–5 yr.	5–17 yr.	1–5 yr.	5–17 yr.	
				%	%		%		%		%
10–20	0										
21–30	2		2	100			50		50		
31–40	38	1	16	42	44	19	3	5	29	0	0
41–50	98	0	30	36	37	21	6	5	27	3	0
51–60	81	1	31	38	37	20	9	2	20	5	7
61–70	70	5	20	29	58	13	7	0	13	3	6
71–80	25	7	9	38	52	32	0	0	12	0	4
81–90	5	4	2	40	60	0	0	0	20	0	20
Total....	319	18	109	34	44	19	6	3	21	3	3

diseases and benign tumors of the breast have but little relationship to malignant disease of the breast; however, in the older age group, past 50, benign tumors may eventually assume local malignant potentialities. In this we refer particularly to intraductal papillomas and fibroadenomas as they occur in the older age group. Pathologically fibroadenomas in this age group can simulate malignancies and care must be exerted by the pathologist to be certain that a malignant interpretation is not rendered on a bizarre fibroadenoma in the older breast.

The management of malignancies of the breast, either by radical or other procedure, is no assurance of the length of survival of the patient, whether metastasis has or has not occurred. It depends rather on the host resistance of the individual. Radical surgery and postoperative therapy with anticancer compounds and irradiation, today offer the only curative procedures we have. We must make an effort to find other methods of approach in conjunction with radical surgery, if we are to improve our statistics of cures.

(From our studies and research, non-neoplastic and benign tumors are not precursors to malignant diseases, and therefore, malignant diseases occur from causes that are as yet unknown and hypothetical.)

Our approach to the treatment of malignant diseases of the breast has had very little change since Halsted's time, with the exceptions of improvement of anesthesia, better understanding of electrolytes and fluid balance, whole blood replacement, irradiation, chemotherapy and hormonal therapy. The efficacy of surgical procedure can only be related statistically, as the patients' responses differ widely. It is, of course, our best approach to malignancy of the breast, but as we all know, it is impossible to positively dissect the patient away from the tumor. This statement is substantiated by Malmgren and associates[3] of the National Institute of Cancer, who reported methods for cytologic detection of tumor cells in the whole blood in 1958, and the article by Malmgren and Potter[4] in the *Southern Medical Journal* in November 1959, reported cancer cells in the circulating blood in patients, before the operation was performed as well as after. This work has been substantiated by Dr. Warren Cole.[1] These investigators also point out, that the demonstration of cancer cells in the blood stream does not always mean that the cancer cells will survive and cause metastatic carcinoma. This is believed to be primarily caused by the host's resistance, which, in later stages of cancer, is lowered, permitting the tumor to metastasize widely.

The prevention of extension and the isolation of malignancy of the breast are undoubtedly best obtained by complete eradication of the breast tissue. At the present time surgery with irradiation probably offers the best means of control. Surgical removal of the ovaries in the premenopausal age group is to be recommended. Chemotherapeutic agents with cytotoxic propensities are certainly of value. The use of isotopes in pleural, peritoneal and bone marrow extensions is likewise of palliative effect. The philosophy of the physician ultimately must be changed to the concept that his task is to make the patient comfortable with the disease rather than to expect a complete cure.

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THE OPERATIVE MANAGEMENT OF ACQUIRED HEART DISEASE

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Any discussion of acquired heart disease must of necessity consider two main categories, the treatment of the cardiac effects of rheumatic fever and the alterations in the coronary arteries secondary to atherosclerotic processes.

It has been said that the surgical manipulation of the heart and the development of techniques for intracardiac operations represent the last great surgical frontier. Whether or not this is true, the motivating force that stimulated these advances was the desire to offer mechanical correction for the valvular deformities that rheumatic fever produces. Current management of rheumatic fever has favorably altered the degree of valvular damage the disease formerly produced. Nevertheless, 100 per cent of those persons who have rheumatic fever will be left with some heart damage, and of these a significant number will have enough damage to shorten their lives or seriously impair their ability to earn a living.

In 1923, Cutler and associates[5] opened a stenotic mitral valve through a transventricular approach in a patient who initially experienced relief of his symptoms, but who succumbed shortly thereafter of surgically induced mitral regurgitation. Subsequent to this initial work, operative attempts at correction of valvular deformities were so discouraging as to preclude for 24 years any serious attempt at solution of the technical problems involved.

In 1948, utilizing the adjuncts of endotracheal anesthesia, blood bank availability of whole blood and the diagnostic and physiologic information afforded by right heart catheterization, Bailey,[1] Harken,[6] and Baker and co-workers[3] separately described the successful finger fracture operation for mitral stenosis. To elaborate on the subsequent acceptance and efficacy of this procedure is no longer necessary. It suffices to say that of those persons with an isolated mitral stenosis who are operated upon because of the incapacitating nature of their disease, 85 per cent will be objectively so improved as to be able to return to normal or near normal activity. The 15 per cent who are not improved, made worse, or succumb, deserve additional evaluation. This group has been carefully studied and several important facts have been elicited which will aid in the future selection of candidates for the closed operation, and those who will require the more elaborate, direct vision approach to their mitral valves.

The earlier concepts of mitral valvular disease were restricted to the proposition that fusion of the commissures was the essential problem, and that lysis of the leaflet edges to the annulus would restore normal valvular function. Unfortunately, subvalvular stenosis involving shortening and thickening of the chordae tendineae is a frequent accompaniment of commissural fusion and commissurotomy results in the development of mitral regurgitation (fig. 1). The failure to recognize the latter situation preoperatively, despite left and right heart catheterizations, dye-dilution curves, and physical findings, occurs not infrequently. The resultant mitral regurgitation cannot be effectively corrected by any closed cardiotomy technique. Before evolution of direct vision evaluation of the regurgitant valve, this group was almost always made worse by blind mitral valvotomy.

The patient with long standing, tight mitral stenosis first develops pulmonary plethora, then pulmonary hypertension, then pulmonary arteriolar sclerosis, and finally right ventricular failure. All of these sequelae to impeded pulmonary venous return are either reversible or correctable except pulmonary arteriolar sclerosis. When this point is reached, the pulmonary hypertension is necessary to allow alveolar capillary gas exchange, and lowering the pulmonary vascular pressure by mitral valvotomy may result in respiratory acidosis and further right heart strain, ending in pulmonary edema. A further sequela of protracted mitral stenosis is thickening of the atrial walls and narrowing of the atrial chamber to such an extent that opening of the mitral valve is not

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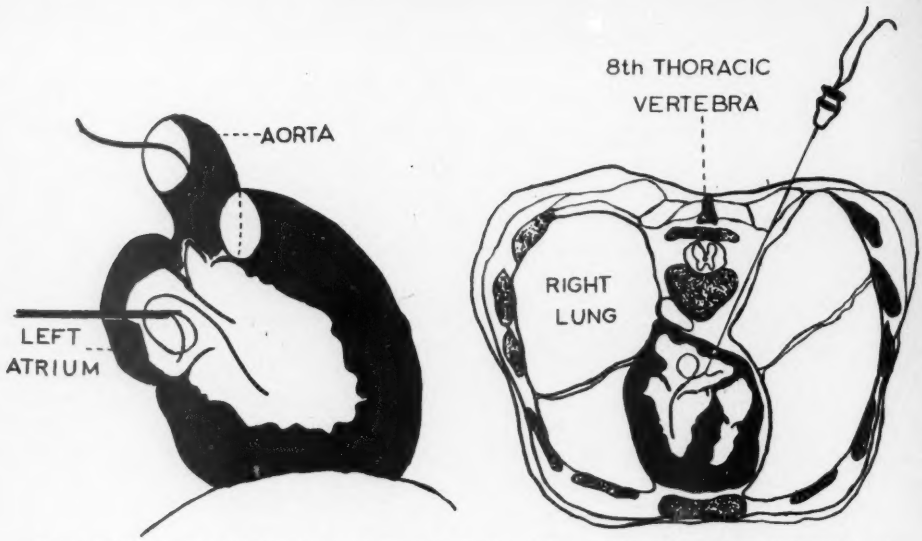

FIG. 1. Technique of left heart catheterization. Catheter in aorta is passed through brachial artery to measure pressure gradient across aortic valve. Note catheter in left atrium for similar determination across mitral valve.

going to result in a significant increase of atrioventricular flow.

Of the estimated 5000 mitral commissurotomies done in the past 12 years, a not insignificant number have progressively developed a recurrence of signs and symptoms that first made them candidates for operative intervention. A small percentage of these have had an exacerbation of their rheumatic fever with additional myocardial and valvular damage. However, the larger percentage had an inadequate operation initially. The anterolateral commissure is easily accessible to the intra-atrial finger, but the posterior medial commissure, where the greatest amount of valvular damage, fibrosis and calcification takes place, is not so readily approached. It is probably inadequate separation of fused posteromedial leaflets that has necessitated the recent rash of "re-do" commissurotomies.

Obviously, the increased understanding of the physiopathology attendant upon mitral valvular disease has altered the indications for operative repair of the stenosed mitral valve, and the newer techniques of cardiopulmonary bypass will decrease the percentage of patients not helped by the operation as originally conceived.

The combination of mitral stenosis and regurgitation requires a surgical philosophy entirely alien to that adequate for pure stenosis. Rheumatic fever affects initially the endocardium. When limited to this superficial cardiac element, valvular fusion is the usual result. However, when the disease in its more virulent forms penetrates the endocardium to involve the deeper layers, fibrosis of the myocardium results. In the valves this is manifested by shrinkage of the leaflets with resultant loss of apposition and regurgitation. Concomitantly, similar involvement of the chordae tendineae with resultant shortening accentuates the regurgitant component. The left ventricular and left atrial enlargement which develops with mitral valvular incompetence further compounds the problem when annular dilation produces further separation of the leaflets.

Since replacement of leaflet tissue by autogenous graft or prosthetic appliances has proven relatively unsuccessful, at present the most feasible surgical approach to mitral regurgitation is posteromedial annuloplasty (fig. 2). This technique, as described originally by Lillehei[8] and Merendino,[11] requires direct visualization of the mitral valve using the pump-oxygenator. Care must be taken in placing the sutures to

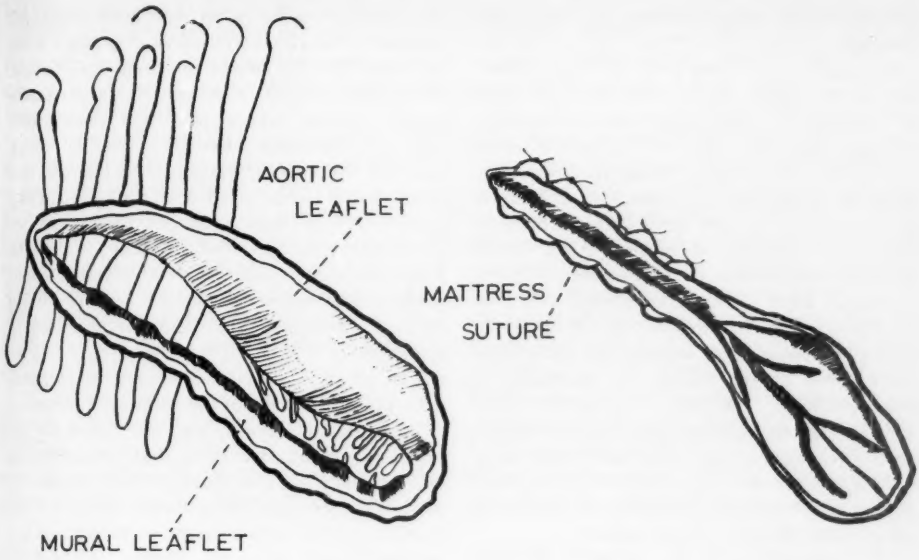

FIG. 2. Method of mitral annuloplasty. Note that sutures are through annulus, and not through leaflets.

avoid the circumflex coronary vessels and the possibility of producing too tight an annulus and mitral stenosis.

In retrospect, it is surprising that the rheumatically damaged aortic valve did not take precedent over the mitral valve in the development of the operative management of rheumatic heart disease. To be sure, the mitral valve is more frequently involved by rheumatic fever, but the sequelae of left ventricular damage are considerably more sinister than are those of right ventricular failure. Perhaps the early appearance of dyspnea and hemoptysis in mitral valvular disease, as opposed to the paucity of symptoms in early aortic valvular involvement, gave precedent to the attack on the mitral valve. It has become apparent, however, that once the left ventricle begins to show evidence of strain, early correction of the valvular deformity becomes mandatory. Syncope and angina in the patient with aortic stenosis precede by only the margin of a few months irreversible failure of the left ventricle and sudden death (table 1).

Historically, Theodore Tuffier in 1912 made the first operative attempt at correction of a stenosed aortic valve through a supravalvular approach. When this operation was unsuccessful further attempts were sporadic and discouraging until 1949 when Bailey approached the stenosed valve through the wall of the left ventricle. Subsequent evaluation of this technique has pointed up the surgical axiom that operative manipulation done blindly is to be avoided if possible. Incomplete separation of the fused commissures with recurrence of the stenosis, or the development of aortic regurgitation have

TABLE 1

*Prognosis of aortic stenosis in 42 patients**

Average age at first observation	53 yr.
Average age at death (39 patients)	55.5 yr.
Mean survival time after onset of symptoms	
Angina	4.7 yr.
Syncope	3.2 yr.
Congestive failure	9 mo.

* Of the 42 patients, 31 were men and 11, women; 14 had a history of rheumatic fever.

deterred most operators from pursuing this technique.

The aortic valve, lying as it does in the direct path of the highest pressure and flow rate areas in the body, is particularly prone to extensive valvular alteration once the protective endocardium is damaged by rheumatic fever. The resultant calcification and complete obliteration of commissural borders have been particularly successful deterrents to restoration of leaflet pliability and valvular competence. Bypass techniques allow direct supravalvular access to the diseased area, but frequent loss of dissection planes between calcium deposits and underlying valve tissue has prevented the restoration of good pliability, and not infrequently valve damage leads to regurgitation. For these reasons much investigative work has been forthcoming seeking a better operative approach or prosthetic devices which can be substituted for the aortic valve, either stenotic or regurgitant.

The conversion of the stenotic tricuspid aortic valve into a more pliable bicuspid valve, disregarding the previous location of the commissures, has been employed occasionally by the University of Minnesota group with equivocal results. Utilizing the principles of peripheral endarterectomy, Mulder and Winfield[12] have developed a technique for separating the calcific deposits along with the endocardium of the leaflets to restore near normal motility to the valve (fig. 3).

Replacement of the aortic valve with a mechanical device presents so many problems that at first consideration they would appear almost insurmountable. Any material that would withstand the countless openings and closings required without fatigue or malfunction would be admirable, indeed. The problem of placing the appliance in such a position as to prevent regurgitation and yet permit coronary artery filling seems formidable, and the prevention of thrombus formation, blood trauma, and embolic phenomena compound the difficulty. However, the helix spring valve of Hufnagel and associates[7] and the "toilet-seat" flap valve of Lillehei[9] would appear to be major advances directed toward ultimate solution.

Of equal importance with the development of operative methods for repair of acquired aortic valvular disease, is the need for accuracy in evaluating the necessity of such intervention. The high mortality rates associated with all methods of aortic valve surgery require that the operation be limited to those individuals whose cardiac status is so impaired as to make further medical management more hazardous than the operations presently available. Data assessing the pressure gradient between the left ventricle and aorta by left heart catheterization, calculations of cardiac output, aortic valve size, and regurgitant blood volume, plus data from the more commonly employed clinical examinations must be carefully evaluated by the medical-surgical team before recommending operation.

The effective surgical treatment of atherosclerotic involvement of the coronary arteries is of such recent vintage, and the series so far reported so small, that an accurate evaluation is not feasible. Beck's[4] classic contributions of pericardial poudrage and coronary sinus ligation, plus his concept of the electric instability of the ischemic heart, need no further elaboration. The Vineberg[13] operation for implanting the internal mammary artery in the ischemic myocardium also is well known. The fact that none of these procedures has been shown to routinely increase the amount of blood available to the myocardium has caused other investigators to seek different methods of dealing with an increasingly serious disease. The fact that one out of

FIG. 3. Mulder technique of aortic valvular endarterectomy. Great care must be exercised to avoid damaging leaflets.

every two persons in the United States will die of some form of heart disease, and that all forms of heart disease except coronary artery disease are on the decline, is impetus enough to encourage these investigations.

Coronary artery atherosclerosis differs from that occurring in the periphery in that it is segmental in its distribution. Fortunately, from the surgical point of view, the segments involved are usually the superficial or visual segments of the vessels. The most common site of involvement is the first portion of the anterior descending coronary artery with involvement of the proximal portion of the right coronary artery next most common (fig. 4).

In light of the increasing acceptance and efficacy of the surgical management of peripheral vascular occlusive disease, it is natural that similar procedures be considered for coronary atherosclerosis. At the present time, Bailey and co-workers,[2] and Longmire and associates[10] are employing thromboendarterectomy in carefully selected patients suffering from coronary occlusion with encouraging results.

The fact that segmental ischemia of the myocardium is particularly conducive to myocardial irritability and ventricular fibrillation, necessitates that certain fairly rigid criteria be met before recommending patients for this operation. Patients with a healed infarct without angina or those in heart failure are obviously excluded. As presently conceived, the operation should be offered only to those patients with angina who have widespread and marked S-T segment depression during an attack and who show no localized S-T segment elevation or QRS alteration, and in whom coronary angiocardiography can demonstrate complete occlusion of a major coronary vessel. Utilizing these criteria, plus the cooperative comanagement of the car-

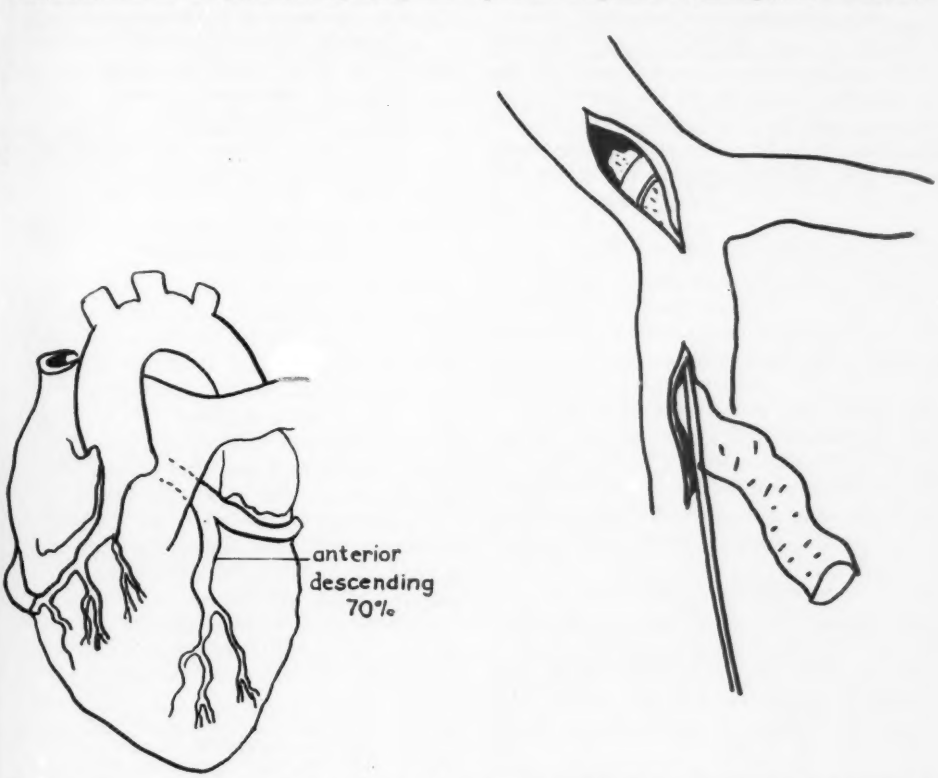

FIG. 4. Common sites of segmental coronary atherosclerosis, and method of coronary endarterectomy

diologist and surgeon both pre- and postoperatively, the means of relieving pain and fear and the return to a productive life may be ahead for a great segment of our aging population.

SUMMARY

Acquired heart disease is becoming, with rapidly increasing frequency, a surgical problem. As presently managed, the mortality of all surgical procedures, except commissurotomy for mitral stenosis, requires careful evaluation preoperatively before recommending operative intervention. The presently most acceptable operations are considered and their advantages and shortcomings discussed.

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NEW CONCEPTS IN PRIMARY HYPERPARATHYROIDISM

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Primary hyperparathyroidism constitutes a challenging question in medicine and surgery. The basic pathogenesis of this condition is not conclusive,[2] and problem exist in diagnosis and management.[14, 20]

This paper is based upon an analysis of all medical, surgical and autopsy cases of primary hyperparathyroidism encountered from 1948 through 1958 (a period of 11 years) in all hospitals in Houston, Texas. There are 38 proven cases of primary hyperparathyroidism discussed. Reference also is made to an additional 13 cases diagnosed during 1959.

The purpose of this paper is to present data and views concerning 3 main problems of primary hyperparathyroidism: (1) an explanation of the clinical types, (2) screening of patients with primary hyperparathyroidism; a new method, and (3) the microscopic adenomas of the parathyroid glands.

1. AN EXPLANATION OF THE CLINICAL TYPES

What Determines the Clinical Type of Primary Hyperparathyroidism?

The clinical findings in primary hyperparathyroidism are variable. In general, 4 clinical types are considered: (1) the pure renal type, manifested by renal lithiasis, nephrocalcinosis and hypercalciuria; (2) the pure skeletal type, manifested by bone demineralization, bone cysts, and osteitis fibrosa cystica; (3) the mixed type, which may be divided (a) into the renoskeletal type, mainly renal with early skeletal changes and (b) the skeletorenal type, mainly skeletal with early renal changes; and, (4) the incidental type, in which signs and symptoms are lacking.

The relative incidence of the clinical types has varied in different reports (table 1). Satisfactory explanations have not been given for such clinical variations. The question remains: *Why does the same disease manifest itself in different clinical pictures?*

Collip and Clark[10] assumed that the primary action of the parathyroid hormone was on the bone. Albright and Reifenstein[1] adopted the view that the action of the parathyroid hormone was excretion of phosphorus through the kidneys with secondary changes in the bone. Later, several authors[38] postulated multiple sites of action of the hormone on both the kidneys and the bones, simultaneously. Finally, the possibility of the presence of more than 1 hormone, each affecting a system in the body, was suggested.[34]

In our series, 2 patients were asymptomatic. The study of 36 patients with symptoms revealed significant points which illuminate the nature of the condition. Certain factors pertaining to the disease and to the patient seem to influence the clinical picture. They are: (1) the disease factors: (a) duration and (b) severity; and (2) the patient factors: (a) age and sex, (b) activity-diet group and (c) previous disease.

The Duration of the Disease

Information from table 2 indicates that the skeletal type usually is diagnosed earlier than the renal type. The mixed types, probably are diagnosed later, and the renoskeletal type, in particular, is of long duration.

The duration of the disease will influence the clinical type in only a few instances. If left untreated for a long period of time, some of the renal and skeletal cases will become mixed types. Others, however, may maintain their pure renal or pure skeletal forms as long as 15 years.

The Severity of the Disease

Because it is not possible to determine the level of circulating parathormone, the level of serum calcium may be indicative of the severity of the disease. The mean level of serum calcium showed variations with the clinical types (table 3).

The skeletal and skeletorenal types show a level of serum calcium relatively higher than

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TABLE 1
Clinical types of primary hyperparathyroidism (Relative Incidence)

Series	No. of Cases	Renal	Skeletal	Mixed
		%		
Norris[32]	314	5.5	60.5	32.5
Hellstrom[18]	56	?35.0	37.4	?25.0
Black and Zimmer[4]	207	65.0	13.5	17.0
Bogdonoff and others[5]	27	45.0	7.5	33.6
Houston, Texas	38	47.2	15.8	31.7

TABLE 2
Duration of primary hyperparathyroidism

Clinical Type	No. of Cases	Average Duration
		mo.
Skeletal	6	30
Renal	18	54
Mixed	12	
Skeletorenal (7)		42
Renoskeletal (5)		151

TABLE 3
*Variation of serum calcium level in primary hyperparathyroidism**

Clinical Type	Mean Serum Calcium Level
	mg./100 cc.
Skeletal	14.8
Renal	13.0
Mixed	
Skeletorenal	14.6
Renoskeletal	12.1
All types	13.5

* Houston, Texas, 38 patients

that of the renal and renoskeletal types. It may be assumed that the disease in the skeletal and skeletorenal types is of greater severity than that in the renal and renoskeletal types. This may explain in part the shorter duration of the disease in the skeletal type than in the renal type and the prolonged duration of the disease in the renoskeletal type.

Age and Sex

The age-sex incidence is illustrated in figure 1. In men, both renal and skeletal types appear during the fourth decade of life. In women, although the renal types appear about the fifth decade, the skeletal types are more common in the sixth decade. Interestingly, all cases of the skeletal types in women over the age of 50 years exhibited postmenopausal osteoporosis which may be considered as a predisposing factor to skeletal affection by hyperparathyroidism.

Activity and Diet

Besides age and sex, the activity and diet of a patient reflect his physical characteristics. The patients were classified into 3 activity groups. (1) the very active, *i.e.*, manual workers; (2) the moderately active, *i.e.*, school teachers and other professional types; and (3) the inactive, *i.e.*, people with physical handicaps.

The clinical picture of hyperparathyroidism can be modified considerably by calcium and vitamin D in the diet. Information was obtained directly from the patients concerning the adequacy of calcium and vitamin D intake. The diet of each patient was considered either adequate or inadequate. When information concerning activity and diet were related, 6 activity-diet groups were observed (table 4).

When the patients were considered by the clinical type of their disease (fig. 2), we noticed that the majority of patients with the renal type appeared in group 3, *i.e.*, the moderately active patient with adequate calcium and vitamin D intake. In the mixed types, renoskeletal and skeletorenal, group 4 had the largest number, *i.e.*, the moderately active patient whose intake of calcium and vitamin D was inadequate. Patients with the skeletal type are usually in group 6, *i.e.*, the inactive patient with inadequate calcium and vitamin D intake.

These clinical findings can be explained by a new concept.[7, 37] The primary effect of hyperparathyroidism is bone dissolution with calcium mobilization. Calcium mobilized from bone is excreted mainly through the kidneys. The clinical picture is the product of calcium metabolism in the body with primary changes in bone and secondary changes in the kidneys.

If calcium absorption is adequate in the active person, the bone has the capacity to compensate the calcium it has lost; hence, bone changes will not appear. When the bone's capacity to assimilate calcium is reduced, as in the inactive person

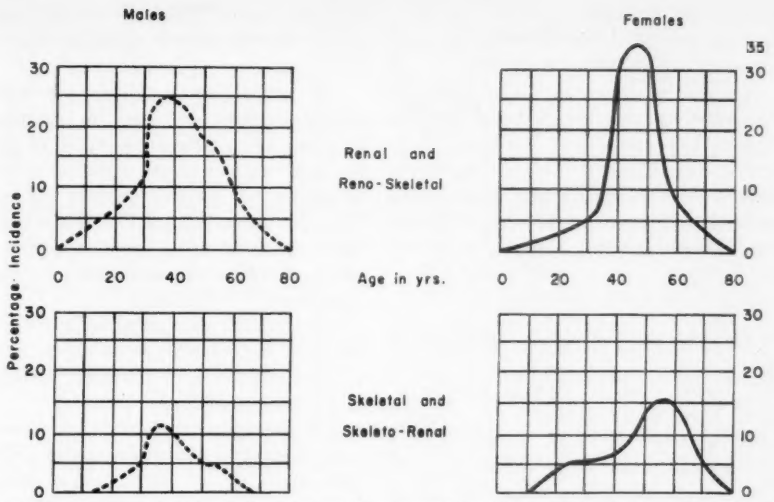

FIG. 1. Age and sex incidence in primary hyperparathyroidism (series in Houston, Texas)

TABLE 4
*Activity-diet groups**

Group	Activity	Diet	No. of Patients
1	Very active	Adequate	0
2	Very active	Inadequate	1
3	Moderately active	Adequate	16
4	Moderately active	Inadequate	13
5	Inactive	Adequate	3
6	Inactive	Inadequate	3

* Houston, Texas, 36 patients.

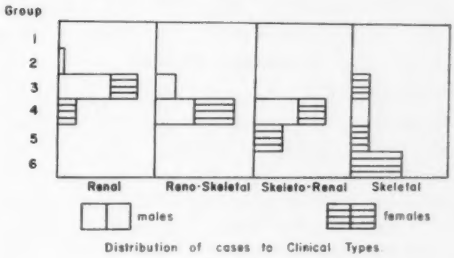

FIG. 2

or in one in whom the calcium absorption is inadequate, skeletal manifestations will develop.

If the kidneys fail to excrete the calcium load, as in excessive calcium intake, or in excessive calcium mobilization from bone, calcium deposits appear in the renal tissue or in the urinary tract with other renal manifestations.

This concept is in line with the latest views on bone metabolism,[9, 12, 21, 23, 24, 29, 30, 33] and recent findings concerning radioactive calcium.[6, 36, 42]

Predisposition to Disease

Predisposition of the skeletal or renal systems to previous disease seems to be an important factor in determining early the system or systems affected by hyperparathyroidism. Forty-five per cent of patients with bone manifestations showed additional osteoporosis which was believed to be present prior to the onset of hyperparathyroidism. Similarly, 40 per cent of patients with renal manifestations gave a history of previous renal disease.

2. SCREENING OF PATIENTS WITH PRIMARY HYPERPARATHYROIDISM: A NEW METHOD

The commonly used tests for screening patients with possible primary hyperparathyroidism include: (1) total serum calcium level, (2) serum phosphorus level and (3) serum alkaline phosphatase level.

Hypercalcemia is considered the best diagnostic aid.[3] Although the level of total serum calcium at which hyperparathyroidism may be suspected has not been established, total serum calcium is

TABLE 5

Total serum calcium levels at which hyperparathyroidism may be suspected

Author	Year	Total Serum Calcium Level
		mg./100 cc.
Black,[3] Mayo Clinic	1953	10.5
Keating,[22] Mayo Clinic	1956	10.3
Cope,[11] Massachusetts General Hospital	1960	10.0

used to estimate the degree of hypercalcemia (table 5).

Several proved cases of primary hyperparathyroidism have been reported, the patients demonstrating levels of total serum calcium below the normal range.[28, 35] In our series of 38 proved cases, 2 patients had average total serum calcium levels of 10.6 mg. per 100 cc., and 1 patient had an average total serum calcium level of 9.3 mg. per 100 cc.

Ionic Serum Calcium

It has been suggested that ionic serum calcium increases in hyperparathyroidism;[25, 26] whereas, the fraction bound to proteins varies with the level of plasma proteins. The ionic calcium could be determined by projecting total serum calcium and total proteins in the nomogram of McLean and Hastings.[26]

When our series was projected in this nomogram, 8 patients showed levels of ionic calcium within the normal range. The ionic calcium is not a true reflection of the degree of calcemia.

Diffusible Serum Calcium

Recently, it was found that the diffusible serum calcium rather than the ionic, more accurately reflects the degree of calcemia.[19, 30, 39, 40] Diffusible serum calcium consists of the ionic calcium and calcium complexes; citrate, carbonate and phosphate. The diffusible fraction of serum calcium dialyzes through the cell membrane. It is considered the biologically active component of total serum calcium.[30] A constant exchange occurs between the diffusible calcium of the serum and the calcium of bone. Because this exchange is affected by the parathyroid glands, the activity of these glands can be most accurately reflected by the level of the diffusible serum calcium. The average level of diffusible serum calcium is 6.6 mg. per 100 cc. This level will serve as a critical line for determining the degree of calcemia.

The techniques available for determining diffusible serum calcium are complicated. A standard and a practical method for general use has not been developed; however, we were able to devise an indirect method for estimating diffusible serum calcium.[17] This method is based upon the relationship between serum calcium, total and diffusible, plasma albumin and plasma globulins[19, 30, 40] (fig. 3).

A new triside nomogram has been developed to estimate the diffusible serum calcium level from the levels of total serum calcium, plasma albumin and plasma globulins.

The nomogram has been tested experimentally and clinically. Diffusible serum calcium was determined by an ultrafiltration method[31] and estimated from the nomogram in 79 persons used as controls. The results were identical in the majority of instances and discrepancies existed in only 4 cases. Also, in all 51 patients with primary hyperparathyroidism, the diffusible calcium as estimated from the nomogram was above the normal level of 6.6 mg per 100 cc. This method serves as a practical screening test for primary hyperparathyroidism.

3. THE MICROSCOPIC ADENOMAS OF THE PARATHYROID GLANDS

Primary hyperparathyroidism is a clinical diagnosis which is substantiated by the presence of hyperfunctioning parathyroid tissue.[16] The only objective of surgical treatment is the removal of the abnormal parathyroid tissue. The recognition of such parathyroid tissue is difficult in certain instances.

Abnormal parathyroid tissue assumes a variety of sizes and shapes which can be recognized by comparison to a parathyroid body of normal size and shape.[8, 13, 15, 27, 41] A normal parathyroid body is 1.5 cm. or less in greater diameter.[13] The average parathyroid adenoma measures 3 to 4 cm. in length, 1.5 to 2.5 cm. in width, and 1.0 to 2.5 cm. in thickness and has a mean volume of 4 cc.* If a parathyroid adenoma attains such a small size and simulates a normal parathyroid gland, it may be impossible to recognize it grossly.

* These measurements were estimated from our series as well as from Norris's collective review.[32]

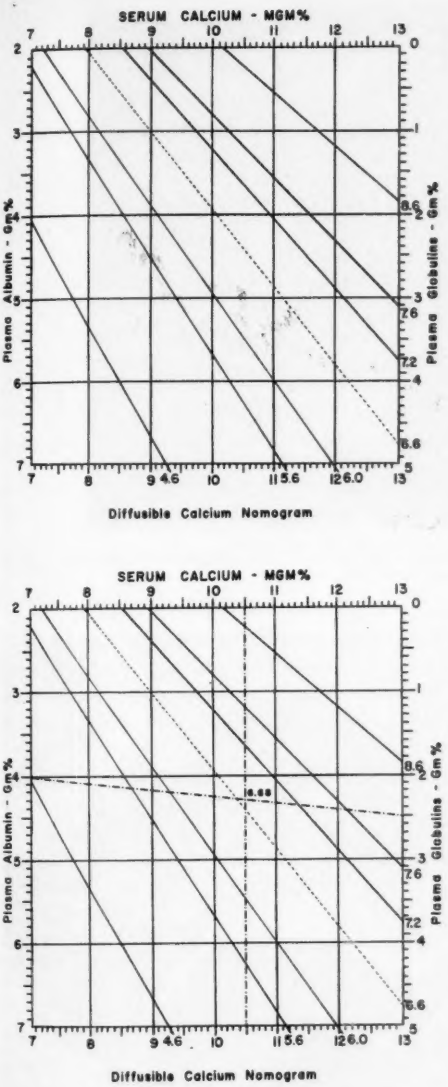

FIG. 3. Diffusible serum calcium nomogram. Application of the nomogram: The vertical scale on the *left* represents the values for plasma albumin, that on the *right*, the plasma globulins. The horizontal scale, *above*, represents the total serum calcium values. A straight line may be drawn between any given values of albumin and globulins. Where a perpendicular line drawn from the total serum calcium value intersects the straight line between the plasma proteins, this point represents the level of diffusible serum calcium. This value is determined by the relationship between the point of intersection and the neighboring oblique lines.

In this series, a total of 40 specimens of abnormal parathyroid tissue were removed; 36 of these were parathyroid adenomas Six adenomas, or 17 per cent, were below 1.5 cm. in greatest diameter. Because abnormal parathyroid tissue could not be distinguished grossly, each entire gland was removed for microscopic examination. The diagnosis was not established until the permanent slides were examined and laboratory studies were obtained postoperatively.

The final pathologic description distinguishes these two types of adenomas: (1) diffuse increase of parathyroid tissue throughout the specimen; 3 adenomas; and (2) within a normal parathyroid gland, a sharply circumscribed nodule of compact parathyroid cells which may represent an adenoma; 3 adenomas. (Frozen sections of these 3 adenomas were studied and were reported "negative" for pathologic changes.)

Figures 4 to 6 illustrate the second type of adenoma. After thorough exploration of the neck, 4 parathyroid glands were identified. The left superior parathyroid gland was larger than the rest, measuring 1.0 by 0.4 by 0.4 cm. This enlarged gland was removed *in toto*, and upon microscopic examination of the section of frozen tissue, only normal parathyroid tissue was recognized. On examination of the permanent tissue section, however, the diagnosis was made. Postoperative evaluation of the patient revealed the disappearance of hyperparathyroidism. Clinical or biological signs of hyperparathyroidism were not present immediately or after 18 months postoperatively.

The detection of such very small or microscopic adenomas is possible if the surgeon considers this entity. Confirmation of the clinical diagnosis before the surgical exploration is essential.

At operation, identification of all parathyroid tissue is important. In 80 to 88 per cent of patients, 4 parathyroid glands can be identified. In 4 to 6 per cent, more than 4 glands are present; and in 14 to 16 per cent, only 3 parathyroid glands are present.[13, 41] Hence, in any exploration of the parathyroid glands, a minimum of 3 parathyroid bodies should be identified. When 3 or more parathyroid glands are identified, the most enlarged gland can be removed safely and should be submitted for microscopic examination.

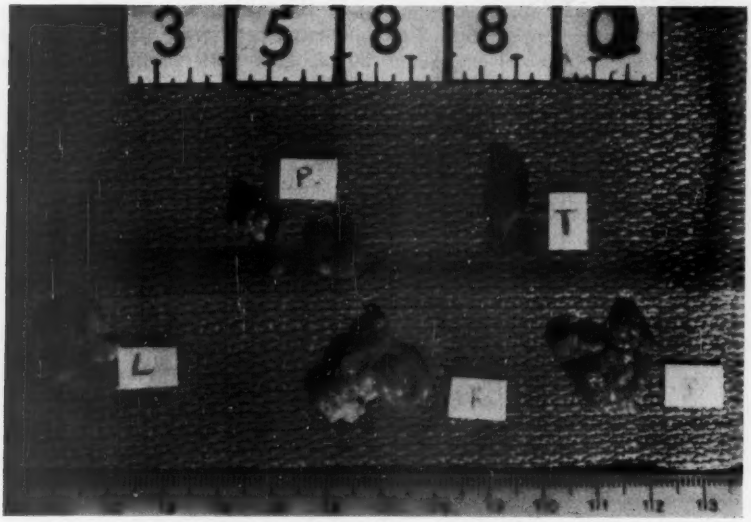

FIG. 4. Photograph of tissue removed in parathyroid exploration. *P*, enlarged left superior parathyroid gland and fat; *L*, lymph node; *T*, thyroid nodule; *F*, fat.

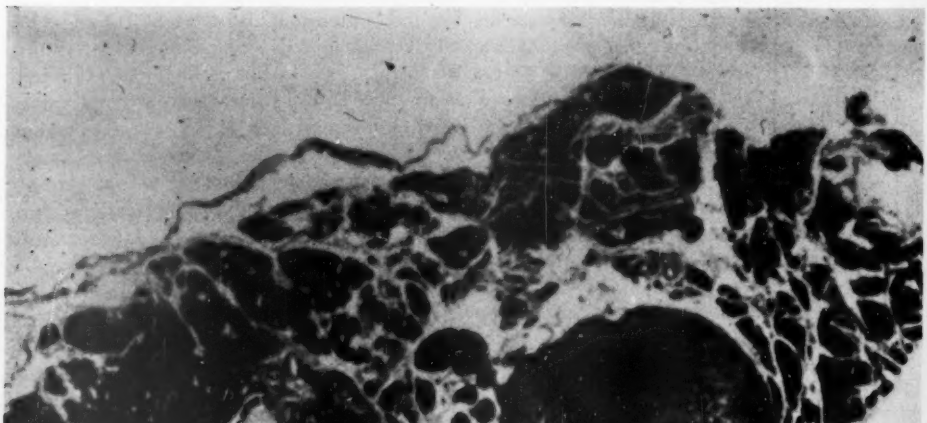

FIG. 5. Permanent tissue section of left superior parathyroid gland (scanning power × 10)

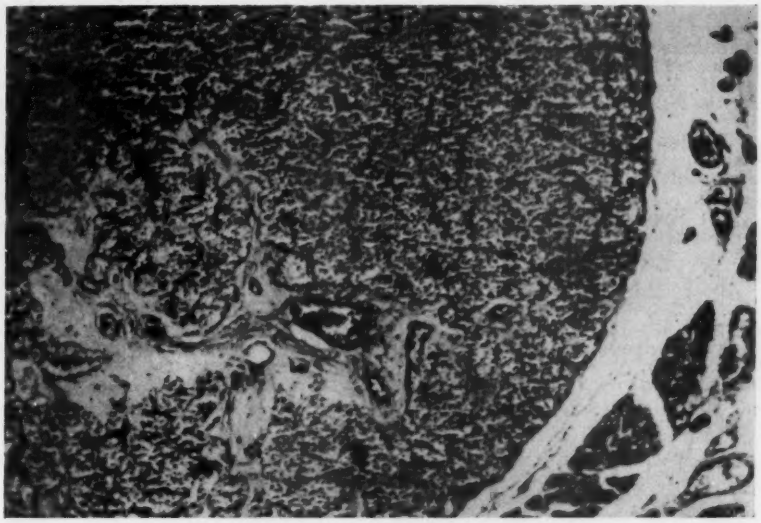

FIG. 6. Same tissue as in figure 5 (under low power)

SUMMARY

Four clinical types of primary hyperparathyroidism are presented: the pure renal, the pure skeletal, the mixed and the incidental types.

In the discussion and review of theories of primary hyperparathyroidism, certain factors pertaining to the disease (duration and severity) and to the patient (age, sex, activity-diet group, and previous disease of the skeletal or renal systems) were found to determine the clinical picture. The explanation offered is in line with that proposed in the most recent investigations.

The commonly used tests for screening patients with possible primary hyperparathyroidism are serum calcium, serum phosphorus, and serum alkaline phosphatase levels. Determination of the serum calcium level is the most diagnostic aid.

When the values of total serum calcium, ionic serum calcium and diffusible serum calcium are compared in the screening of patients, diffusible serum calcium is found the most reliable. A new and a practical method is suggested for estimating diffusible serum calcium.

Parathyroid adenomas may be microscopic in size and may be difficult to recognize during surgical exploration. A policy of identifying 3 or more parathyroid glands during the surgical procedure is very helpful, and may be the only means of detecting such microscopic adenomas.

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LIFE-ENDANGERING FORMS OF ENDOMETRIOSIS

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Endometriosis, as we know, is a fairly common disease occurring in relatively young women who are in the childbearing age. It is not usually considered a disease which is serious insofar as the life of the patient is concerned. The main purpose of this presentation is to bring to your attention a few cases where the life of the patient was actually threatened due to complications arising from this usually benign condition.

Endometriosis can be classified as being internal (adenomyosis) or external. The external variety is usually the type which concerns us more, as the lesions produced by this form have been found to involve the ovaries, bladder, cul-de-sac, appendix, small and large bowel, lymph nodes, vulva, vagina, perineum, groins, arms, umbilicus, hernial sacs and abdominal incisions.

ETIOLOGY

The exact etiology of endometriosis lends itself to a great deal of controversy. Sampson's[20] well known theory, that endometrial tissue can extrude from the tubes in a retrograde manner during menstruation, remain viable and grow on tissues in the peritoneal cavity, still has many advocates and is probably the most widely accepted. This theory has been further strengthened by the animal experiments of Te Linde and Scott[23] who produced endometriosis in monkeys, experimentally, by having them menstruate into the peritoneal cavity. Sampson's theory is also further strengthened by the reports of several other authors[4, 8, 17, 21] who have observed endometriosis occurring in the vulva, perineum or vagina following surgical trauma. It is strongly felt by these observers that menstrual blood contains viable endometrium, capable of growing, if the right medium presents itself. These observations are somewhat in opposition to the Novak[16] theory of coelomic origin of aberrant endometrium.

Javert[7] in 1949, proposed a "composite theory" to include not only direct extension, exfoliation and implantation, but also lymphatic and hematogenous metastasis. This would explain endometriosis being present in lymph nodes in the pelvis, and also its apparent metastasis to the lung as reported by Nicholson[14] in 1951.

Numerous other theories have been advanced, most of them to be discarded due to lack of sufficient evidence and to the high degree of theoretical postulation. The exact etiology of endometriosis still remains somewhat in doubt. It has been observed by several authors, including Weed[24] and Meigs,[11] that women who get pregnant early in life are not inclined to develop the disease. This probably accounts for the low incidence of endometriosis in the lower strata of our society where early pregnancies are the rule.

CASE REPORTS

Cases of endometriosis involving the lower colon and producing bowel obstructions of varying degrees have been reported by numerous authors.[1, 9, 10, 18, 22, 26, 27] In my survey of the literature, there were no cases reported where an endometrioma of the sigmoid colon had perforated, producing the drastic clinical picture of a large bowel perforation.

The first patient to be reported, a 44-year-old white woman, was admitted to Sparks Memorial Hospital at 2:30 a.m. on July 30, 1952. She stated that 2 days before admission, she had noted a gastrointestinal upset, and that at 6 a.m. the day before admission, she awoke with lower abdominal cramping pain which became increasingly severe. She then became nauseated and vomited. She vomited several times during the day, and was seen by her local physician who stated that she had hyperactive peristalsis. Later that night her abdominal pain became more severe; she became faint, and her local physician could not obtain her pulse or blood pressure. Glucose was given, and she was rushed to the hospital by ambulance. Further questioning disclosed the fact that she had occasionally passed blood per rectum since 1942, but there had been no definite change in her bowel habits. Her menstrual history was normal. She had had no previous abdominal operations or pregnancies. Physical examination revealed an acutely ill woman. Her blood pressure was 100/80,

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and her pulse was 140. She appeared to be in a moderage degree of shock. The abdomen was distended, bowel sounds were absent, and there was a considerable amount of muscle guarding with tenderness to deep palpation and rebound tenderness in the lower abdomen. There appeared to be a fullness in both lower abdominal quadrants. Pelvic examination disclosed the uterus to be pushed down by what appeared to be a pelvic mass. On rectal examination a liquid, reddish stool, characteristic of blood, was noted. The red blood count was 4,210,000 with 14 gm. of hemoglobin. The white count was 9900 with 77 segs., 16 stabs, 6 lymphs and 1 monocyte. A flat plate of her abdomen revealed findings strongly suggestive of a mechanical obstruction within the lower small bowel loops. She was prepared for surgery as quickly as possible with a preoperative diagnosis of possible mesenteric vascular occlusion being made. Upon opening the abdomen, there was found approximately 250 cc. or more of cloudy, purulent fluid in the peritoneal cavity, and both the small and large bowel showed marked evidence of a peritonitis. There was evident an annular lesion of the sigmoid, appearing grossly to be a carcinoma, which had perforated. The perforation was a fairly small one, but it had produced a considerable amount of contamination. In both adnexal areas there were cystic masses, each about the size of an orange. The perforation was sutured, the pelvic cavity was drained, and a transverse loop colostomy was then performed. The patient's condition during the entire procedure was precarious, and her blood pressure had to be maintained with Levophed and blood. The postoperative clinical diagnosis was carcinoma of the sigmoid colon with perforation. The lesion in the sigmoid was resected 3 weeks after her first surgery. At the time of this procedure it was found that she had an abscess extending up the left gutter, which had perforated into the splenic flexure of the large bowel, necessitating a wide resection of the left colon. The left ovary, which was the size of an orange and contained chocolate material, was removed. A catheter cecostomy was done. Following this surgery, she made an uneventful recovery. The final microscopic diagnosis proved the annular lesion of the sigmoid to be an endometrioma with perforation (fig. 1). The left ovary showed endometriosis. This patient returned later for further surgery in 1956 because of menorrhagia, and a hysterectomy was performed for multiple uterine fibroids. At this time she also had a moderate sized serous cyst of the right ovary, and, microscopically, adenomyosis of the uterus.

The next case is one of massive internal bleeding, occurring in a rather unusual manner, due to endometriosis. In my survey of the literature, I have been unable to find any other such case. This patient, a 32-year-old white woman, the mother of one child, was admitted to Sparks Memorial Hospital on December 23, 1958. Her history showed that she had been ill for the past 2 or 3 days with lower abdominal cramping. On the morning of admission her condition suddenly became critical, and she was seen by her local physician. He told her that she was "bleeding internally," and rushed her to the hospital. Her history disclosed that she was 2 weeks overdue on her period, but that she had not had any abnormal bleeding. She stated that she felt very weak and "worn-out." When first examined she appeared to be exsanguinated. Her pulse was barely perceptible, and her blood pressure, when first taken, was not obtainable. The abdominal examination revealed fullness and tenderness to palpation in the lower abdomen. On pelvic examination the uterus seemed to be somewhat enlarged, and a possible adnexal mass was felt on the left side. A diagnosis of ruptured ectopic pregnancy was made, and the patient was immediately prepared for surgery. Her hematocrit on admission was 23 per cent, and she had 8.0 gm. of hemoglobin. Her white blood count was 29,100 with 81 segs., 2 stabs, 1 eosinophile, 11 lymphocytes and 5 monocytes. At the time of surgery, it was conservatively estimated that there was at least between 2000 and 2500 cc. of fresh blood in the peritoneal cavity. The point of bleeding was occurring from a small ruptured endometrial cyst, which had eroded into the right cornual artery of the uterus, and active bleeding was still occurring from this rather good sized vessel. The endometrial cyst was excised, and the area sutured to stop the hemorrhage. The left ovary showed definite evidence of endometriosis. Following the surgery, the patient made an uneventful recovery, and left the hospital in 7 days. Microscopically, the excised lesion proved to be endometriosis (fig. 2).

Malignancy developing in endometriosis is a fairly rare situation. Dockerty and co-workers[3] have reported adenocarcinoma of the rectovaginal septum, probably arising from endometriosis. Adenoacanthoma arising in ovarian endometriosis has been reported by several authors.[2, 12, 15, 25] Malignant stromal endometriosis has been reported by Hunter,[5] among others. In most instances, the malignancies reported are adenocarcinomas. This next case represents adenocarcinoma originating from ovarian endometriosis.

This patient, a 32-year-old white woman, was admitted to the hospital on December 10, 1957, with a known history of a pelvic tumor for at least the past 6 or 7 years. Her recent menstrual history revealed the fact that she had been having her

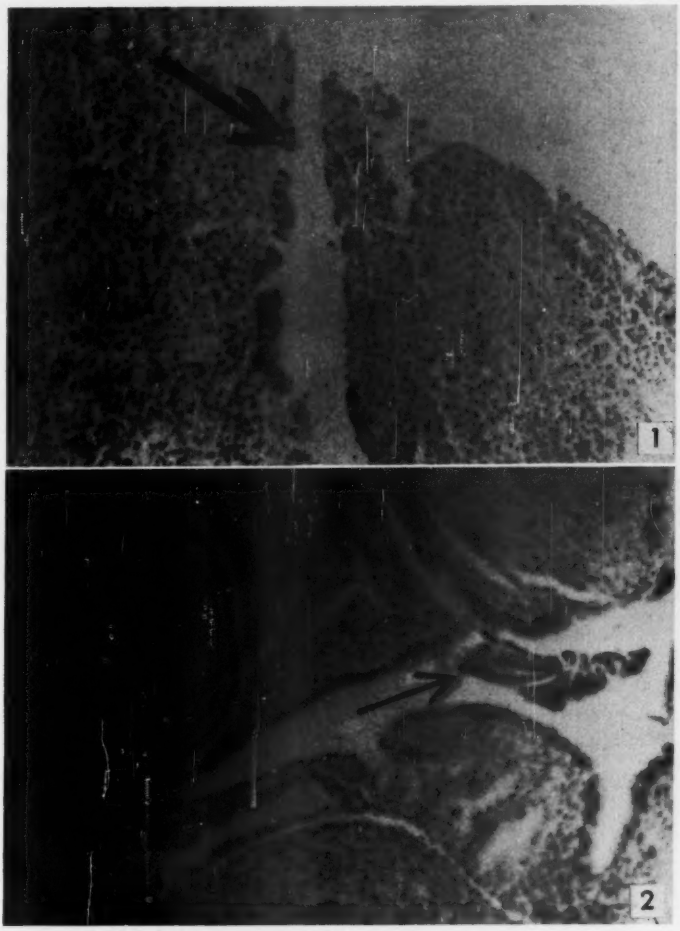

FIG. 1. Shows ulceration of the mucosa of the large bowel and endometrial epithelium (*arrow*)
FIG. 2. Showing endometriosis in region of cornual artery

periods up to 2 weeks late, and having some intermenstrual discharge. She had recently noted some aching in both of her legs, and about a week previously, had noted the onset of pain in her left calf. Her history was otherwise without interest, except for the fact that she had urinary frequency. Pelvic examination confirmed the presence of a large pelvic tumor, presenting more on the left side, and feeling rather cystic. There was also felt to be a smaller tumor on the right side. It was felt that the pain in her leg was due to nerve pressure. A preoperative diagnosis of bilateral cystic ovarian masses was made, and the patient was prepared for surgery. At the time of exploration, she was found to have a large grapefruit-sized left ovarian cyst, containing chocolate material. On the right side she had a similar cyst, about one-half as large. These cysts were adherent to the pelvic cavity, and dissection was difficult because of numerous adhesions. The uterus grossly felt normal. The appendix showed evidence of chronic inflammation. A bilateral salpingo-oophorectomy, total hysterectomy, and an appendectomy were performed. A considerable amount of difficulty was encountered in the dissection because of the dense adhesions to adjacent viscera. The patient made an uneventful recovery after surgery. Microscopic examination of both cysts revealed endometriosis, but in the right ovary there was found to be frank adenocarcinoma, microscopically com-

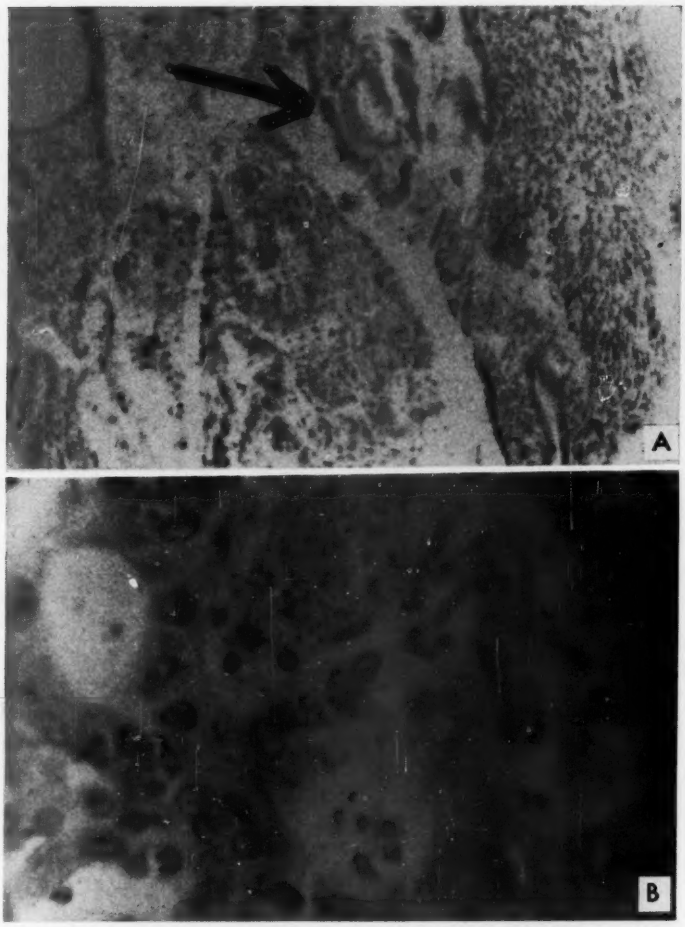

FIG. 3A. Showing carcinomatous change in endometriosis of right ovary (*arrow*)
FIG. 3B. High power magnification of endometrial adenocarcinoma developing in endometriosis

patible with that of an adenocarcinoma of the endometrium. The final diagnosis proved to be endometriosis involving both ovaries, the right tube and the appendix, with endometrial adenocarcinoma arising from endometriosis involving he right ovary (fig. 3*A* and 3*B*). It was felt that her prognosis was poor due to apparent seeding of the peritoneal cavity. This patient was given a full course of cobalt^{60} therapy. She has been followed closely now for 2 years, and has shown no evidence of recurrent malignancy.

SUMMARY

1. Although endometriosis is usually considered a relatively benign disease, there are situations where the life of the patient may actually be threatened.

2. Three cases have been presented to illustrate this point. These include: (a) a case of perforation of an endometrioma of the sigmoid colon; (b) a case of massive internal bleeding due to erosion of a fairly large vessel (the cornual artery) by an endometrial cyst; and (c) a case of adenocarcinoma developing in endometriosis involving the ovary.

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NONTUBERCULOUS PSOAS ABSCESS[a]

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Nontuberculous psoas abscess is an important clinical entity which must be understood by the surgeon and the practitioner. It is an extraperitoneal infection, most commonly infecting young males and in general is characterized by a uniformity of findings.

This subject is briefly discussed in the standard texts and literature. Our interest was recently stimulated by two simultaneous cases at the Crippled Children's Memorial Hospital in Oklahoma City, Oklahoma. These two cases and four others are reported in table 4.

Long[1] in 1924, described this condition as a syndrome of nontuberculous psoas abscess, characterized by flexion deformity of the thigh, tender mass of recent formation above the outer portion of Poupart's ligament, moderate pain about the inguinocrural region, remittent fever, elevated leukocyte count and emaciation and weakness.

ANATOMY

The psoas muscle has its origin from the transverse processes and bodies of the 12th thoracic and all the lumbar vertebrae (fig. 1). It courses over the superior ramus of the pubis and under Poupart's ligament to insert into the lesser trochanter of the femur. Posterior medially lies the spinal column and the quadratus lumborum muscle. Anteriorly lies the peritoneum. Superiorly the psoas muscle passes beneath the arcuate ligament of the diaphragm. It is in close relationship with the kidney, pancreas and small intestine. Through it runs the lumbar plexus. Anterior to the muscle lies a maze of iliac lymph nodes. Actually there are three chains of nodes: the mesenteric, the celiac and the lumbar. The drainage of these nodes is important in considering the causes of the abscess (table 1).

ETIOLOGY

It is believed that there are four common methods of infection. These are suppurative lymphadenitis, direct extension from an abscess or areas of osteomyelitis, hematogenous spread and trauma.

Suppurative lymphadenitis. An infection of any of the areas drained by the iliac nodes (mesenteric, celiac or lumbar) could rupture into the muscle mass. The lumbar chain drains the lower extremity as well as the male and female reproductive organs, the descending colon and the sigmoid. We believe that this chain is the most common offender. Examples of this category would be furuncles, osteomyelitis and infected hollow or solid viscera.

Direct extension of an abscess. This could originate from an empyema of the lung, or perinephritic abscess, or any other abscess which breaks into the retroperitoneal space. Osteomyelitis is included in this type of etiology and its extension to the psoas muscle is most common from the spine or pelvis.

Hematogenous spread. Hematogenous massive bacteremia may lodge groups of pathogens into the psoas muscle with resultant abscess formation.

Trauma. A sprain or tear of the psoas muscle leads to formation of a hematoma. This then may be followed by infiltration of the hematoma by microorganisms of the circulating blood to bring about the formation of an abscess.

COURSE OF THE ABSCESS

Generally the abscess follows the path of least resistance; this is down toward the insertion of the muscle. It may point just medial to the psoas, or it may enter the adductor triangle and point to the upper third of the thigh. Many variations

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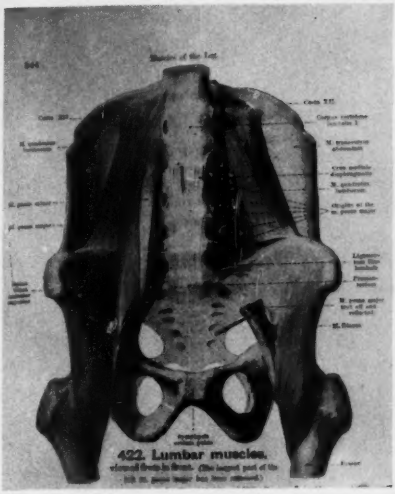

FIG. 1. Anatomy of the iliopsoas muscle (from *Atlas of Human Anatomy*[7]).

TABLE 1

Chain of Nodes	Drains the Following Systems
Mesenteric	Small intestine, ascending colon transverse colon
Celiac	Stomach, liver, pancreas
Lumbar	Lower extremities, male and female reproductive organs, descending colon, sigmoid, rectum

can occur, but most commonly the abscess points anteriorly, generally above the outer portion of Poupart's ligament.

SYMPTOMS AND PHYSICAL FINDINGS

The history of the process varies. It may be either acute or chronic at onset. In the chronic type the patient may have been sick for as long as 4 weeks with the onset of incipient progressive characteristic findings. In the more common acute phase the patient may be very toxic and have an elevated temperature and pronounced pain in the lower back and anterior hip regions. Flexion contracture of the thigh, between 150 and 90°, is a characteristic finding. This particular finding probably leads to the diagnosis more than any other. The patient may have such a severe hip flexion contracture, with secondary lordosis, that his walking is a grotesque sight (fig. 2).

FIG. 2. Stance of patient with iliopsoas abscess, illustrating severe lordosis and hip flexion contracture.

FIG. 3. Roentgenogram of the lumbar spine and pelvis, illustrating the bulge of the iliopsoas muscle on the left.

It is of great importance to note whether with sedation and gentle manipulation the patient will allow the hip to be fully extended, because many disease processes about the hip are relieved of pain by flexing the femur.

Examination of the hip reveals that pain is not as severe with increased flexion, abduction, adduction or external rotation. However, internal rotation, and particularly extension of the hip, stretches the psoas muscle, thereby producing severe pain. There may be a tender mass which can be palpated or an abscess may be presenting itself, generally above Poupart's ligament or about other sites previously mentioned.

TABLE 2

Differential diagnosis of nontuberculous abscess

Infected hip joint	Iliopsoas burstiis
Arthritis of the hip	Trauma to the spine
Traumatic synovitis of the hip	Tumor of the spine
Tuberculosis of the hip	Renal or perirenal abscess
Osteomyelitis of the pelvis and spine	Abdominal abscess
Arthritis of the sacroiliac	Reproductive organs
	Solid or hollow viscera

ROENTGENOGRAMS

Generally the survey of the spine and pelvis is negative except for the possibility of osteomyelitis of the pelvis. The presence of an abscess producing a disturbance of the normal psoas shadow is seen in a small number of cases. Zadek[3] reported one out of seven which produced a protrusion of this shadow (fig. 3). An intravenous pyelogram may show the kidney displaced upward and the ureter medial to the psoas shadow; it is an important diagnostic procedure.

LABORATORY

There is a marked elevation of the leukocyte count up to 15,000 or 20,000, with a marked shift to the left (80 to 90 per cent) of polys. This is associated with an elevated sedimentation rate. There may be a positive culture of staphylococcus or streptococcus from the blood stream.

DIFFERENTIAL DIAGNOSIS

A very careful differential diagnosis must be made prior to onset of treatment. Table 2 presents some possibilities which should be carefully considered. In general the history, the physical examination, the x-rays and the laboratory findings help to make a differential diagnosis.

One of the most important differentials is that

TABLE 3

Differential diagnosis of tuberculous and nontuberculous psoas abscess

Findings	Nontuberculous Abscess	Tuberculous Abscess
White blood count	Elevation	Normal
X-ray of chest	Negative for tuberculosis	May be positive for diagnosis of tuberculosis
Family history and clinical contacts	Negative for tuberculosis	Strongly positive
Evidence of chronic illness	Rarely seen	Almost always
Calcification of abscess	Negative	Positive
Relationship of abscess to Poupart's ligament	Generally above	Generally below
History of trauma to lower extremities	Of great importance	Not important
Importance of recent infection of gastrointestinal tract, reproductive organs or lower extremities	Of great significance	Of some importance
Clinical appearance of abscess	"Hot"	"Cold"
Remittent fever	Yes	No
Flexion contracture of thigh	Present	Almost never present
Cough; night sweats	Negative	Positive
History of osteomyelitis	Of great significance	Of no significance

TABLE 4

Review of 6 new cases of nontuberculous psoas abscess

Patient	Age	Sex	Race	Symptoms	Physical	Clinical Course	Culture
EJD	21	M	W	Fever and malaise, 2 wk., followed by pain right hip; inability to extend because of pain	Abdomen tender, right lower quadrant; severe pain on extension; tender groin	Surgical drainage gave dramatic relief as did sensitive antibiotics	Hemolytic *Staphylococcus aureus* Coagulase-positive, tuberculosis-negative
FJ	2	F	W	Pain, left hip; fever and malaise, 3 wk., followed by inability to straighten hip	Acutely ill; left thigh flexed to 80°; pain on any extension; acute swelling, left thigh	Course critical until drainage of 1500 cc. pus; high dosage of penicillin daily	Hemolytic *S. aureus* Coagulase-positive, actinmyosis + tuberculosis - negative
RJG	19	M	W	Chronic osteomyelitis right ilium; pain in groin, worse on extension, becoming so severe he walked with hip flexed; low grade fever, 2 wk.	Hip kept flexed at all times due to pain; other motions not painful; abdomen tender, right lower quadrant	Surgical drainage gave immediate relief of symptoms; penicillin given	Not recorded
FB	20	F	N	Pain, swelling, left thigh, 2 yr.; malaise, weight loss; low grade fever	Marked swelling, left hip to ankle; fluctuation in femoral triangle; pain on extension.	Surgical drainage and antibiotics produced dramatic relief	Gram positive cocci
BJJ	11	M	N	Pain left hip 10 days prior to admission; low grade fever, malaise, loss of appetite; flexion deformity left hip; marked lordosis; tender right lower quadrant; unable to extend past 120°	Abdomen tender right lower quadrant; unable to extend past 120° due to pain; flexion and rotation not painful	Surgical drainage and antibiotics daily; immediate subsiding of fever and progressive improvement in hip function; full extension in 1 week	Hemolytic *S. aureus* Coagulase-negative
LA	5	M	W	Fever, vomiting and abdominal pain, 5 days	Marked muscle spasm, right hip and thigh; hip flexed to 110°; refused to bear weight on right leg; voluntary rigidity right lower quadrant with tenderness; unable to extend past 110° due to pain	Surgical drainage and antibiotics; dramatic improvement; slight restriction of extension at 1 mo.; asymptomatic, incision well healed	Hemolytic *S. aureus* Coagulase-positive

of the tuberculous psoas abscess (table 3). A clinical history pertaining to contacts and family relations with tuberculosis, loss of weight, night sweats, hemoptysis and chest pain are also important to consider before making a decision. Roentgenograms of the chest, calcifications within the abscess and clinical evidence of tuberculosis of the lung are other important diagnostic points. Physical findings of a nontuberculous abscess are rarely present in an abscess produced by tuberculosis.

TREATMENT AND CLINICAL FINDINGS

In the event of doubt about the presence of an abscess, a needle aspiration can be performed. However, the possibility of introducing infection into the peritoneal cavity must be kept in mind.

Many incisions have been described, including (1) McBurney's, (2) an incision just above Poupart's ligament posteriorly or (3) an incision over the crest of the ilium laterally. All are aimed to stay outside the peritoneal space. There may or may not be evidence of recent hemorrhage. The abscess may vary in size but will sometimes yield up to 600 or 700 ml. or more of purulent material. It is recommended that a drain be left in place until the heavy drainage has subsided. It can then be gradually withdrawn. Antibiotics should be started as soon as the abscess is diagnosed. Cultures and sensitivity tests should be taken at the time of surgery. Most of the cases reviewed were caused by streptococcus or staphylococcus.

PROGNOSIS

A dramatic response is seen following surgery. The flexed thigh slowly returns to normal position; sometimes gentle traction may be necessary to accomplish this. The fever subsides along with the pain, spasm and tenderness.

In 6 new cases presented (table 4) and in 20 cases reviewed in the literature and all treated by surgical drainage, there were no deaths reported.

SUMMARY

1. Nontuberculous psoas abscess is a clinical entity which needs the understanding of both the surgeon and the practitioner.

2. The anatomy, etiology, symptoms, x-rays, laboratory, differential diagnosis, treatment and prognosis are discussed.

3. We believe that the condition most commonly is due to a rupture of an iliac node into the muscle.

4. The most common course of the abscess is to point above the outer portion of Poupart's ligament.

5. The most common characteristic finding is a flexion contracture of the thigh, associated with signs of an abscess mass in the lower anterior abdominal quadrant.

6. Most commonly there is not a protrusion of the psoas shadow on the roentgenograms; however, an intravenous pyelogram may show a displacement of the kidney or ureter.

7. The most common disease to be considered in a differential diagnosis is a tuberculous psoas arising from tuberculosis of the spine.

8. Treatment is surgical drainage and with this the prognosis is excellent.

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VESICAL NECK OBSTRUCTIONS IN CHILDREN

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The purpose of this paper is to call attention to the prevalence of congenital vesical neck obstructions and to emphasize the role such obstructions play in causing recurrent urinary infections and kidney damage. It will be pointed out that in many instances, irreparable renal damage occurs, especially in those children for whom a proper urologic examination is delayed while the patient is treated for recurring bouts of urinary infection. It is not good practice to administer urinary antiseptics to children with recurrent or persistent pyuria without proper urologic investigation. In children with vesical neck obstruction, delay in adequate diagnosis has often resulted in damage to bladder and kidneys beyond repair. Congenital obstruction at the bladder neck occurs a great deal more often than is commonly believed. The probable etiology is faulty embryologic development. Multiple anomalies are often encountered in association with vesical neck obstructions. The complex embryology of the genitourinary tract no doubt can be blamed for the high incidence of such anomalies.

Congenital obstructing lesions at the vesical neck are usually valves or bar formations in boys and contractures in girls. Other types of obstruction at the vesical neck seen in boys may be due to an enlarged or hypertrophic verumontanum, and in some instances hypertrophy of the muscle fibers at the vesical neck (figs. 1 and 2). Patients with vesical neck obstructions may be grouped, I, II, III, according to the severity of the obstruction and also according to the extent of dilation damage to the urinary system. Evaluation of each patient is important as to the degree of obstruction and damage. Proper management of the patient is dependent upon this evaluation.

Patients with mild obstructions may have symptoms that are quite troublesome and yet may have only minimal damage to the urinary system. A large percentage of enuretic patients and those with frequency and urgency are in this group I with mild obstructions and minimal urinary tract damage.

A survey of 368 children with bladder dysfunction revealed that 314, or 85 per cent had urethral or vesical neck pathology. These 368 children exhibited symptoms of frequency, dysuria, urgency, enuresis, dribbling of urine and feeling of incomplete emptying of the bladder as the major complaints.

Careful urologic examination was done on each patient and in 326 the degree of obstruction and urinary tract damage was found to be grade I or II. In 42 patients or slightly more than 10 per cent the degree of urinary tract damage was severe and was considered to be grade III. With obstruction at the vesical outlet the back pressure changes in the upper urinary tract are bilateral as a rule, but may be more marked on one side than the other. The bladder is also damaged by this back pressure. In the presence of obstruction at the vesical neck or in the urethra, the increased intracystic pressure causes hypertrophy of the bladder muscle and trabeculations in the bladder wall. This pressure increase is gradually transmitted against the ureterovesical valve until, in many instances, the valve mechanism is no longer effective, and vesicoureteral reflux or regurgitation takes place. Such back flow up the ureters causes ureteral dilation and ureteral stasis with urinary infection. The most important and vital consideration of this increased pressure in the ureters is that such urinary back pressure causes damage to the kidney, even in the absence of complicating infections. This destruction to renal parenchyma often progresses to such a degree that insufficient kidney function remains for life and health (figs. 3 and 4).

SYMPTOMS

The symptoms of vesical neck obstructions are commonly those of frequency, urgency, enuresis, day or night, dysuria and straining to void. Frequency and urgency are the most common symptoms along with enuresis. Extra urinary

FIG. 1. Drawings depicting various types of valves seen in the posterior urethra in boys with vesical neck obstruction.

tract symptoms often seen are suprapubic soreness, low grade fever, nausea, failure to gain weight and general malaise, loss of appetite and other gastrointestinal symptoms.

DIAGNOSIS

The diagnosis of vesical neck obstruction may be suspected on the basis of symptoms such as enumerated above, but cystourethroscopic examination is needed for confirmation. Present day infant instruments and cystoscopes allow adequate visualization in all ages of both sexes. Cystograms may be useful to demonstrate ureteral regurgitation, and retrograde pyeloureterograms can visualize the degree of dilation and point out the renal damage. Blood chemistry and renal function studies are useful in confirmation of renal damage.

TREATMENT

Evaluation of the extent of damage is important. This evaluation should include the damage to the ureters and bladder as well as the degree of renal parenchymal damage. In the majority of patients, the damage to the bladder muscle and upper urinary tract has been minimal and treatment consists of correction of the bladder neck obstruction. This may be done by a Y V-type plastic operation (Bradford Young procedure) or by transurethral resection of the bladder neck obstruction. Modern children's resectoscopes allow vesical neck revision under good visualization with less shock and danger than open operations. We (fig. 5), prefer to do transurethral procedures and have obtained excellent results in most instances. When the examination reveals marked dilation of ureters and pelves with vesico-

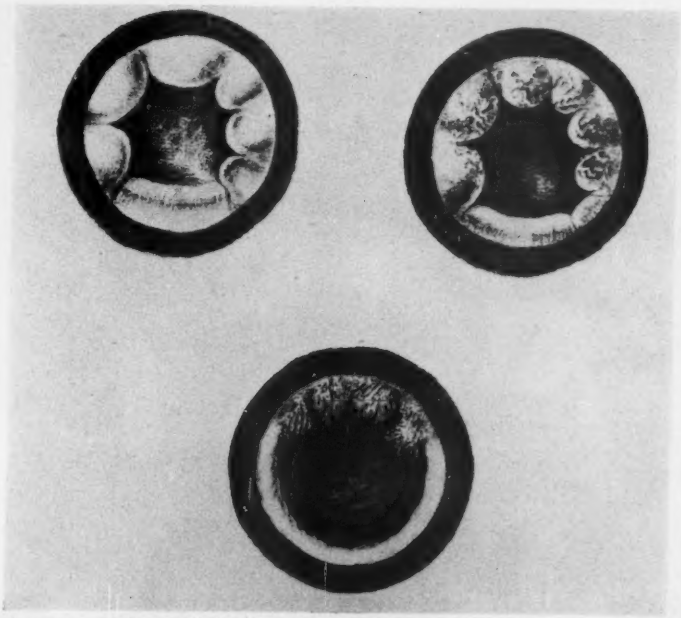

FIG. 2. Drawings of vesical neck obstructions in girls as viewed through forward vision cystourethroscope.

FIG. 3. Urethroscystogram in 4-year-old boy showing vesical neck bar obstruction and trabeculated bladder with diverticulum.

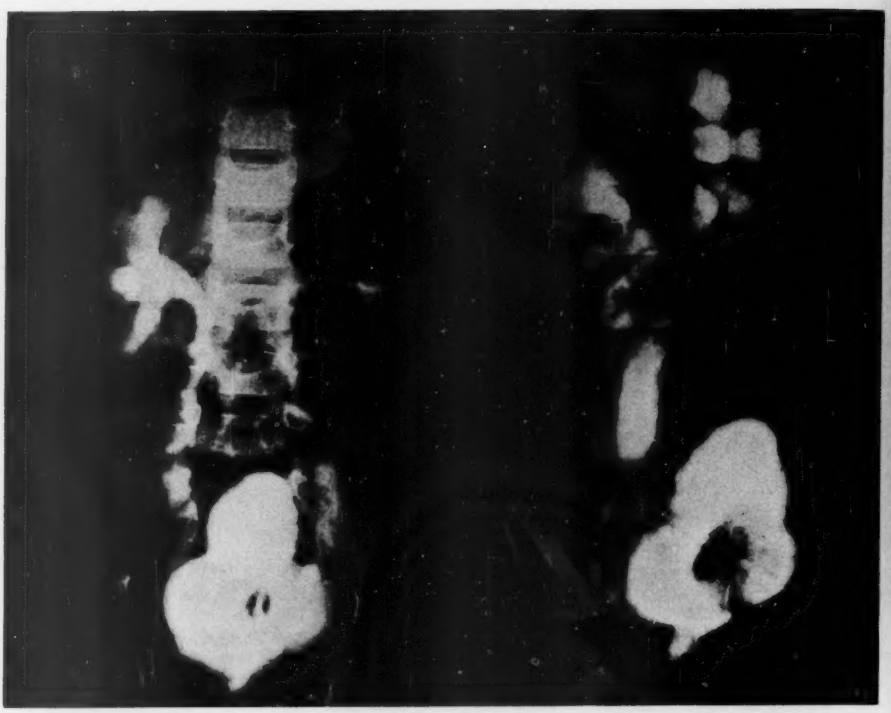

FIG. 4. Pulpo-axial and oblique films showing vesicoureteral reflux with marked renal destruction, due to vesical neck contraction in 5-year-old boy.

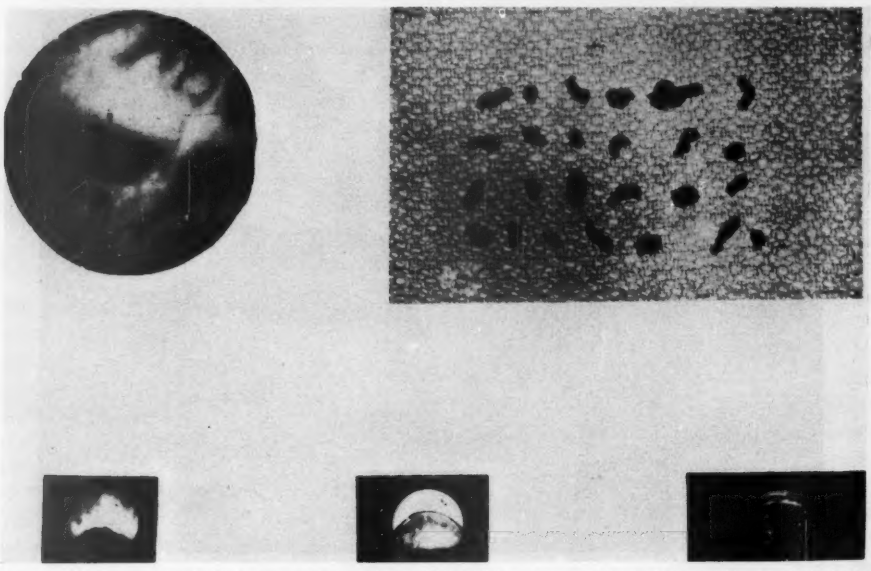

FIG. 5. (*Upper photos*). Showing trabeculations in bladder and resected pieces. (*Lower photos*). *Left*, showing vesical neck obstruction; *center*, resectoscope loop in place; *right*, vesical neck after resection.

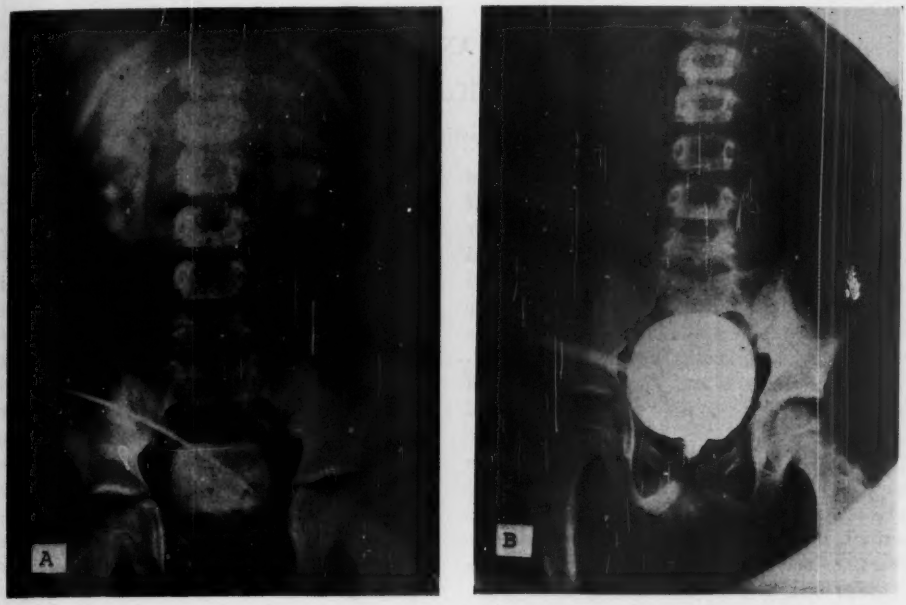

FIG. 6. (*A*) Suprapubic cystogram showing dilated ureters and pelvis with reflux due to congenital bladder neck obstruction. (*B*) Same patient 4 years later, showing absence of reflux; also wide open bladder neck after transurethral resection. Urine clear and general health excellent.

ureteral regurgitation of urine, a series of operative procedures may be needed in order to bring about the optimum result. Long-time suprapubic drainage has been needed and in some instances pyelostomy drainage was necessary to halt the renal damage and control infection and stasis (fig. 6).

Excision of redundant ureter with reimplantation in the bladder to re-establish valve action is needed in some instances.

The poorest results are obtained in those patients with widely dilated ureters with reflux and marked renal destruction. In many instances these patients are seen too late for any real benefit.

SUMMARY

Vesical neck obstructions occur rather frequently in children. The damage to the urinary tract has often progressed to a marked degree before the condition is suspected, and the diagnosis made. Symptoms of frequency, urgency, enuresis, recurrent pyuria, low grade fever, soreness in the lower abdomen over the bladder, loss of appetite, and other gastrointestinal disturbances should suggest the possibility of urethral or vesical neck pathology. Diagnosis is made by proper urologic investigation. Treatment is by transurethral resection of the vesical neck obstruction or open operation on the vesical neck. In patients with marked upper tract damage preliminary drainage and a series of corrective operations are often needed. Patients with seriously damaged renal parenchyma have definitely reduced life expectancy, even though early results may be encouraging. A plea is therefore made for a higher incidence of suspicion of vesical neck pathology in children with recurrent urinary infections and/or symptoms of frequency, urgency, enuresis, dysuria, slow stream, straining to void, low grade fever, soreness or pain over the bladder region, lack of appetite, slow growth or unexplained gastrointestinal symptoms.

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AORTIC LEFT ATRIAL SHUNTS AND PULMONARY HYPERTENSION

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The problem of pulmonary hypertension has proved a challenging one, whether it is a result of primary disease of the pulmonary arteries or cardiac lesion. Its experimental production would provide an opportunity to learn more about its genesis and effects. Furthermore, it should prove of inestimable value in studying possible methods of assessing, and factors determining, reversibility and irreversibility. Thus far, in spite of numerous attempts, no fully satisfactory method has been developed.

Attempts to produce pulmonary hypertension have been generally directed toward procedures which increase the pulmonary arterial blood flow, restrict the pulmonary venous return to the heart, reduce the capacity of the pulmonary vascular bed, or a combination of these procedures. Several groups have studied the effects of production of an end-to-side left subclavian-pulmonary artery anastomosis,[7, 14, 15] or of a side-to-side pulmonary artery-aortic anastomosis,[14, 18] but no elevation of pulmonary arterial pressure was observed following these procedures and no pulmonary vascular changes. Ekström and his associates,[6] however, did obtain pulmonary hypertension in a single animal following an aortic-pulmonary anastomosis. Muller and his colleagues[4, 5, 17] produced pulmonary hypertension and some vascular alterations by means of an end-to-side left pulmonary artery-aortic anastomosis. Few of the animals, however, survived this procedure. Ferguson, his associates and others[9-12] obtained similar results from an end-to-end left subclavian-pulmonary artery anastomosis. Muller's[4, 5] group was more successful in producing pulmonary vascular changes by anastomosing the subclavian artery or the aorta to a left pulmonary lobar artery. The changes, however, were limited to one lobe. Lynn and Bahnson[16] obtained no significant alterations from the production of atrial septal defects by the method of Blalock and Hanlon.[2] The experimental creation of a ventricular septal defect by Griffin and Essex[13] did not result in pulmonary hypertension either. Lynn and Bahnson[16] did produce reversible pulmonary hypertension in some dogs which survived the creation of an atrial septal defect and the production of an artificial ductus arteriosus.

Muller and his associates[17] carried out left subclavian artery-pulmonary vein anastomosis without significant results. Van Bogaert, his associates and others[8, 19, 20] produced transitory elevation of the pulmonary artery pressure, which they felt was of reflex origin, by ligation of the left pulmonary vein. Van Bogaert and his colleagues[21] also studied the effect of anastomosing the aorta to the left atrium and succeeded in producing pulmonary hypertension in 4 of 19 animals; 13 of them died within 3 weeks.

Ferguson and his group obtained vascular changes in some animals subjected to resection of various portions of lung combined with either an end-to-side left subclavian-pulmonary artery or an end-to-end left subclavian-pulmonary lobar artery anastomosis. Moderate elevation of pulmonary artery pressure was noted in 4 of 10 dogs in which Cohn and Magladry[3] carried out an anastomosis between the left subclavian artery and the left pulmonary artery, accompanied by ligation of the left inferior lobar pulmonary artery. Inconsistent results were obtained by Barnes and his colleagues[1] from constricting the pulmonary veins with cellophane bands and subsequent lobectomies.

Altogether, the experimental results reported up to the present leave much to be desired. It is of interest that extensive vascular damage has been observed as early as 2 to 3 months after surgical manipulations by Muller and his associates and by Ferguson and his group on the one hand, but have not developed after 3 years of continued elevation of pulmonary artery pressure in the animals treated by Lynn and Bahnson.

The present study was undertaken in order to determine whether the creation of an aortic-left

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atrial shunt would produce pulmonary hypertension and to study other hemodynamic effects of this procedure.

MATERIALS AND METHODS

Mongrel dogs weighing from 15 to 30 lb. were used. Anesthesia was induced by the intravenous administration of sodium Pentothal. The trachea was intubated and oxygen was administered rhythmically by means of a mechanical respirator. Exposure was obtained by means of a left fourth intercostal incision. The descending thoracic aorta was dissected free, the pericardial sac was opened, and a shunt was created between the aorta and the left atrium by means of a short nylon or Teflon graft. The graft was anastomosed end-to-side to the left atrial appendage with a continuous over and over suture of 5-0 arterial silk. The other end of the graft was then anastomosed to an incision in the aorta with the same suture material, using a curved Potts' vascular clamp for exclusion of a segment of the arterial wall. Blood pressures were measured in the left atrium, in the graft, and in the aorta with the graft open and clamped, utilizing a strain gauge manometee and a Sanborn Twin-Viso cardiette. Each animal was given 200,000 units of penicillin intramuscularly for the first 3 days and were fed a normal diet on the second postoperative day.

At the beginning the operative procedures were carried out without aseptic technique. The incidence of complications, and especially of thrombosis of the graft, was much higher in them than in the others in which sterile technique was utilized.

Second, and in a few instances, third operations were carried out in order to determine the patency of the graft and to measure pressures. Postmortem examinations were carried out on those animals which died or were sacrificed.

RESULTS

There were 40 animals which survived the procedure; 24 died in from 1 day to 5 months after operation. In 11 of them death appeared to be caused by heart failure as the consequence of creating too large a shunt. They all showed pulmonary congestion or edema and dilatation of the heart. We sacrificed 16 animals in from 11 days to over 4 months after operation; 14 of them were sacrificed over 2 months, and 6 of them over 4 months postoperatively.

TABLE 1

Pressure changes in mm. of Hg observed after creation of aortic left atrial shunt

Operative Procedure	Graft Occluded		Graft Open	
	Range	Mean	Range	Mean
Initial				
Left atrium	4–12	6.5	7–18	10.5
	0–6	2.5	1–8	4
Graft			20–85	44
			16–65	33
Aorta	80–150	112	70–140	97
	60–130	94	35–90	69
Second				
Left atrium	3–7	5	7–34	25
	0–3	2		9
Pulmonary artery	8–24	15	10–40	22
	6–16	10	7 –20	12
Aorta	80–140	102	45–120	88
	70–110	86	30–90	60

Pressures were measured at the initial operation in 29 instances (table 1). The pressure in the left atrium rose when the graft was opened and fell when it was temporarily occluded. Mean pressures were 6.5/2.5 with the graft closed and 10.5/4 with it functioning. The aortic pressure fell when the graft was opened. The mean pressure with the graft closed was 112/94 and 97/69 with the graft open. The mean pressure in the graft itself was 44/33.

A second operative procedure was carried out in 19 animals; in 10 from 1 to 2 months, in 5 from 2 to 3, in 3 from 3 to 4, and in 1 over 5 after the initial procedure. At the time of the second exploration the grafts were patent in 6 of 10 animals operated upon with aseptic technique but only in 2 of the 9 operated upon with nonsterile technique. Pressures were measured in the 8 animals with patent, and in 8 with occluded grafts. In those with closed grafts the mean pressures in the left atrium, pulmonary artery and aorta were respectively, 5/2, 15/10 and 102/86. In contrast, they were 29/9, 22/12 and 88/60 in those with open grafts.

In 3 animals a third study was carried out from 3 to 5 months after the initial study. In each the left atrial and pulmonary artery pressures were higher during the final than during the intermediate study, and in each the aortic pressure was higher (table 2).

TABLE 2

Long-term pressure changes in mm. of Hg following aortic-left atrial shunts

Dog No.	Interval after Operation	Left Atrium	Pulmonary Artery	Aorta
	weeks			
748	11	5/2	15/5	95/75
	20	12/6	20/10	110/80
828	6	12/7	16/8	75/35
	15	28/14	40/20	100/75
873	4	14/4	16/10	80/50
	12	20/10	28/15	90/75

In no instance was any evidence of pulmonary vascular sclerosis observed.

DISCUSSION

These experiments constitute a preliminary exploration of the effects of aortic-left atrial shunts. A larger series of animals studied for a longer period of time is necessary in order fully to study the effects of this procedure. These initial studies, however, permit certain conclusions. The majority of the grafts remain open if aseptic technique is used. The size of the graft is of great importance in the survival of the animals. Prostheses larger than ½ the size of the aorta are poorly tolerated. It appears that nylon grafts with a diameter of ¼ in. are well tolerated in small dogs; nylon grafts with a diameter of 5/16 in. and Teflon grafts with a diameter of ¼ in. in dogs of medium size, and 5/16 in. Teflon grafts in large animals. Creation of an aortic-left atrial shunt influences the pressures in left atrium and aorta immediately. A drop occurs in the aortic, and a rise in the atrial pressure. With the passage of time, the animals with patent grafts demonstrate a significant increase in left atrial pressure and a slight increase in pulmonary artery pressure. Those animals studied from 3 to 5 months after the operative procedure show a progressive increase in left atrial and pulmonary artery pressure while the initial depression of aortic pressure tends to correct itself with the passage of time.

During the period of observation, no changes in the pulmonary vessels were observed.

These initial explorations suggest that it may be advisable to study animals with an aortic-left atrial shunt over a longer period of time.

SUMMARY

The creation of an aortic-left atrial shunt tends to bring about a reduction in aortic systolic and diastolic pressure which tends to correct itself with the passage of time. It brings about an increase in left atrial pressure which tends to become progressively more elevated as time passes. There is a tendency for the development of mild to moderate increase in pulmonary artery pressure. No vascular changes in the lungs are noted in animals observed up to 5 months after operation. It would appear advisable to study animals with aortic-left atrial shunts over a longer period of time.

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